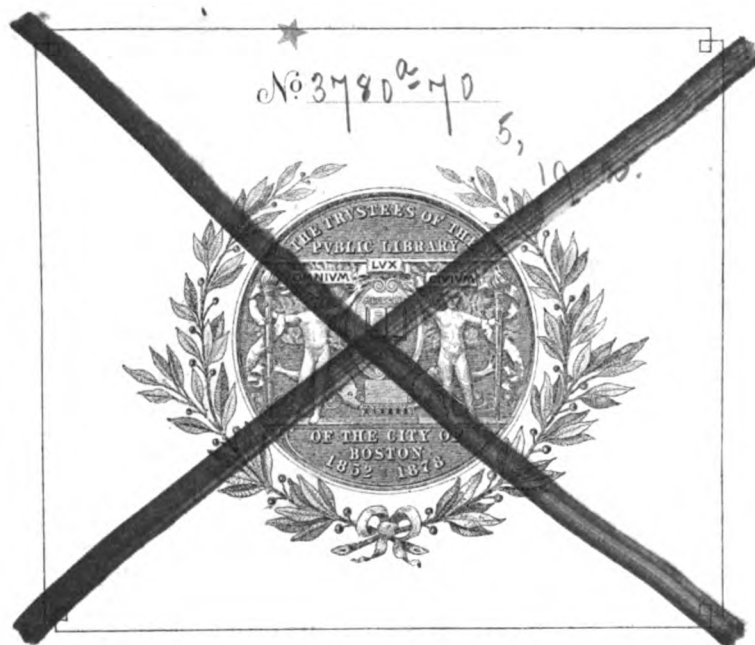




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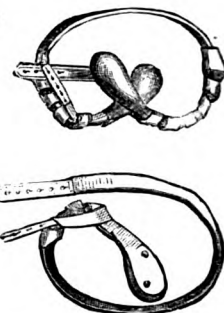
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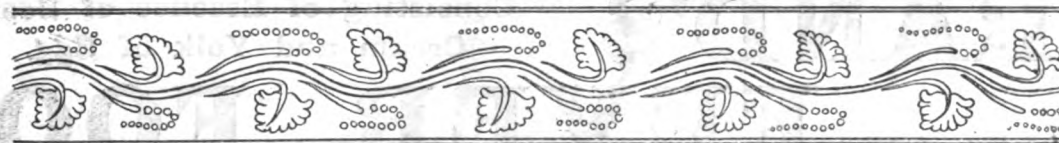
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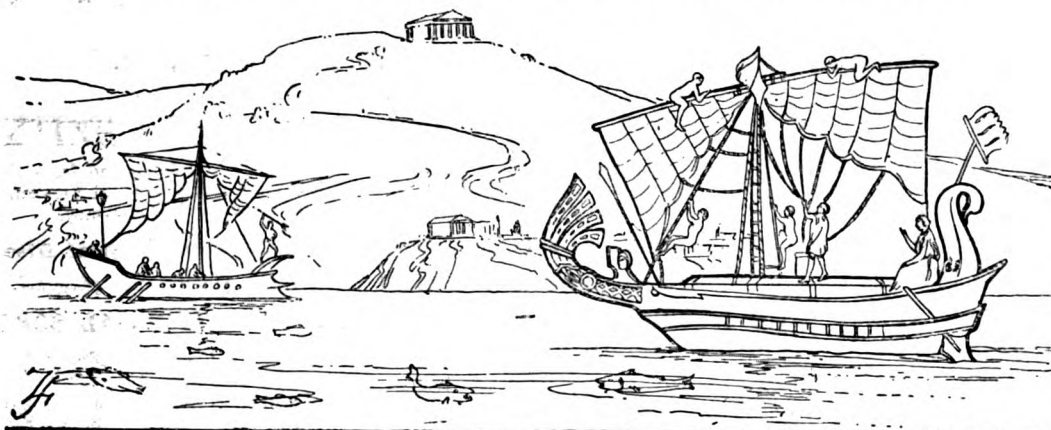
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Original Communications.

SNAKE POISONING IN CENTRAL AFRICA.

By NEIL MACVICAR, M.B., C.M.Edin.

Late Medical Officer to the Church of Scotland Blantyre Mission, British Central Africa.

Case I.—Native man, aged about 35, a tall, slender, active man, was bitten by a snake about four feet long on January 11th, 1899, at about 5 p.m. He killed the snake, but it had bitten him in two places on the back of the right hand. Quickly the hand swelled and became very painful. He walked a little way (to his house) and then lay down. That night he slept well, but during the night he began to pass blood on micturition. All through the act only blood came, and that in large quantity and with little pain. The hand meantime was swollen and bleeding. On the 12th there began to be pain in the epigastrium and both hypochondriac regions. This became so severe that during the night of the 12th he had no sleep. He continued to pass blood. 13th: Was brought to hospital in the forenoon.

State on Admission.—He cannot walk, but this is from weakness. There is no paralysis. He is intelligent and can talk without difficulty. The hand is greatly swollen. The places bitten can be easily seen, they are both on the back of the hand and are about $\frac{3}{4}$ in. apart. At each place there are visible the marks of the fangs—two tiny cuts parallel to each other, and about $\frac{1}{10}$ in. in length. Blood still exudes from the bites and does not coagulate. Examined with the microscope this blood seems normal. The red blood-corpuscles are well formed; the leucocytes are plentiful and are moving freely.

There is constant, severe pain in the epigastrium and in both hypochondria, and on attempting gently to palpate the liver and spleen the pain produced is unbearable. He has eaten nothing since he was bitten.

There is no pain in the region of the bladder. He passes abundant urine mixed with blood. Microscopically examined it is seen to contain numerous red blood-cells, most of them "ghosts," empty of their hæmoglobin. No casts; no ova of Bilharzia; one or two cells of bladder epithelium.

The patient is also coughing and expectorating blood. This, he says, began yesterday.

From the activity of the leucocytes that were emerging from the wound, I judged that the poison was no longer very abundant locally. I therefore merely applied a simple antiseptic dressing. Hot belladonna fomentations were applied round the liver, stomach and spleen.

14th.—Blood count made in forenoon. Result, 5,350,000 red blood-cells per cm. Not a single leucocyte seen in 160 squares. To-day is keeping nothing down. Is constantly vomiting a watery, slightly bilious liquid. (He was not vomiting on admission.) My notes are not explicit on this point, but I think the vomiting began when he attempted to take liquid nourishment after admission on the 13th. 6 p.m.: Has been vomiting all day. Two motions, almost pure blood. Has passed urine five times, looking almost like pure blood. Last time the amount was three ounces. Coughing a good deal, some red blood. Liquid extract of ergot, 1 drm., was kept down a short time and then vomited. 11 p.m.: Pulse 88, regular but flopping, a bad pulse. Temperature 97° 6' (see chart for course of temperature). Pupils normal, react to light. The pain in

SNAKE BITE.—CASE I. TEMPERATURE CHART.

| 1899. Jan. | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 |
|---------------|------|------|-------|------|------|-------|------|-------|-------|----|
| Forenoon . . | 98.2 | 97 | 96 | 98.4 | 97.7 | 100.2 | 98.2 | 99.4 | 97.3 | 97 |
| Afternoon . . | 99.8 | 98.4 | 100.8 | 99.6 | 99.9 | 97 | 99.9 | 100.2 | 100.1 | — |

the liver persists in spite of belladonna fomentations. Has now almost constant hiccough.

I injected 10 cc. of Calmette's serum, which, though

of date 1896, appeared clear and good. Injected slowly under the skin of the right flank. I watched the case closely for about an hour, but could detect no change one way or another.

15th, morning.—Worse. Pulse 140, weak. Has been hiccupping and vomiting all night. Has passed a lot of blood both by bowel and in urine. 3 p.m.: Still hiccupping and vomiting constantly. Can keep nothing down. Pulse cannot be counted, but seems about 140. Whisky hypodermically brought back a countable pulse, 116, but very weak. Condition of patient could not well be more grave.

Thinking that the vomiting might be due to the efforts of the gastric glands to eliminate the poison, or might possibly be uræmic, I decided to try whether assistance could not be rendered to the stomach by the action of the sweat glands. I therefore gave $\frac{1}{2}$ gr. of pilocarpin, and watched anxiously what effect would follow. There occurred slight perspiration, and, I thought, a decided increase in the quantity of the watery vomit. The patient vomited about three pints in half an hour. The pulse remained unaffected.

The blood: Hæmoglobin estimated at between 50 and 60 per cent. of the normal. Microscopically examined: Leucocytes seem very numerous. No count made. The red corpuscles are mostly of uniform size and of normal appearance, but there are a few very small ones.

Blood is still passing by the bowel. Belladonna fomentations over liver continued and poultice over kidneys. Evening: Patient looks very ill. Pulse hardly countable. Still vomiting. Is asking for tea, and thinks he can keep it down.

16th, morning.—During the night patient slept a good deal and vomited only twice. Had tea repeatedly and did not vomit it. This is the first thing he has retained since he was bitten. No motion during the night, but urine still full of blood. Pulse much better—108. Still taking tea in cupfuls. No vomiting, though still some hiccupping. 6 p.m.: Vomiting a little. Urine becoming clear. One motion black and containing some red blood. Complaints of great pain in the epigastrium and up the centre of the chest, probably in the gullet due to retching. Can eat nothing, but takes tea.

17th.—Steadily improving. Able to take flipped eggs and milk. No vomiting. Liver not enlarged in any direction. Still painful.

18th.—Slept well. No vomiting. Pain in liver less severe. Urine quite clear. Pulse 84.

20th.—Quite well except for pain at site of anti-venom injection. Pulse 72, good.

22nd.—Left well.

The snake was said to be the "nsongo" (Yao language) or "mbobo" (Mang'anja language). Unfortunately I could not procure it. This "nsongo" is, however, generally regarded as rather mythical. Natives say of it that "it crows like a cock," but when pressed, they admit that they have never seen it. This patient, however, says quite decidedly that he was bitten by an "nsongo."

Case II.—Native man, aged about 20. On May 29th, 1897, was bitten by a snake in the foot. Snake not seen. Immediately there occurred great pain and

swelling. At night could not sleep on account of the pain, but had no constitutional symptoms. Able to take food well.

30th.—Walked to hospital, about three miles. Pain and cedema now extend up to knee. At the place bitten, on the dorsum of the foot, there is only one scratch, about $\frac{1}{2}$ in. long. I think the snake must have struck obliquely so that one fang missed and the other slanted off, instead of penetrating deeply. The wound seemed so superficial that I simply dressed the foot and leg with a belladonna fomentation.

June 2nd.—Swelling of leg down, but swelling of foot continues, and patient complains of great pain, and thinks a fang is embedded. I cut across the place and found no fang.

3rd.—A brown discharge of altered blood came from the wound to-day. Pain and swelling continue.

6th.—Pain and tenderness at the wound considerable. Swelling gone.

8th.—Pain gone. Some tenderness. Wound almost well. Went home.

11th.—Returned. Wound open again. Pain.

14th.—Wound healed, but still painful. Not seen again.

Case III.—Native man aged about 20. This patient was bitten in the hand with what appears from his description to have been a puff-adder. He says he was very ill with internal pain and with swelling and pain of the whole arm. A fortnight after the bite he came under my care.

The thumb was black and gangrenous and attached to the hand by the bare metacarpal bone and the naked tendons. The whole hand was much swollen and covered with foul sloughs, from beneath which pus exuded. The skin of the back of the hand was dead. The fingers could be moved a little and had some sense of touch.

After removal of the dead thumb and sloughs from the hand, the surface granulated and healed well, the patient retaining the use of his fingers to a slight extent.

Note.—I have seen a number of other cases of snake-bite in natives and one in a European, and all of them were more or less like Case II., local pain and swelling being the chief symptoms. I have never seen a fatal case. My experience has, however, been limited to the Shire Highlands where the snakes are not so venomous as they are said to be in parts of the country that lie at a lower level.

I recollect seeing one case of acute conjunctivitis in an out-patient which was said to have been caused by a "spitting" snake. Having myself seen specimens of this snake (in the possession of Dr. Stephens) I can well understand how the accident might happen. The snake is a small one, and when irritated it squirts a fine shower of venom at its adversary.

Case I., I think is of especial interest. It seems to be the case that in the venom of all poisonous snakes there exists a constituent which has the power of destroying blood-cells (both red blood-corpuscles and leucocytes), and which also acts on the plasma, so that if introduced rapidly (e.g., into a vein) it causes extensive clotting, but if introduced slowly (e.g., subcutaneously) it destroys the normal coagulative power of the blood (Dr. C. J. Martin). It also acts upon the

capillaries, so damaging them that they give way and allow of extravasations (Dr. Weir Mitchell).

This constituent, though present in the poison of all poisonous snakes, is met with only in very small quantity in the poison of most colubrine snakes, while in the poison of Indian vipers it is present in sufficient quantity to produce marked hæmorrhagic symptoms, these symptoms supervening after the patient has recovered from the earlier symptoms of prostration and paralysis (Allbutt's "System of Medicine").

In my case the hæmorrhagic symptoms were of the gravest kind and were of late occurrence, the destructive action of the poison continuing with increasing virulence until the fifth day.

Snake venom is said to be excreted by the kidneys and perhaps also by the salivary glands. This case seemed to suggest that the gastric glands were performing a similar function.

Case III. illustrates the well-known tendency of snake wounds to cause extensive suppuration, the consequence, no doubt, both of the loss of germicidal power of the serum and of the septic condition of the snake's teeth.

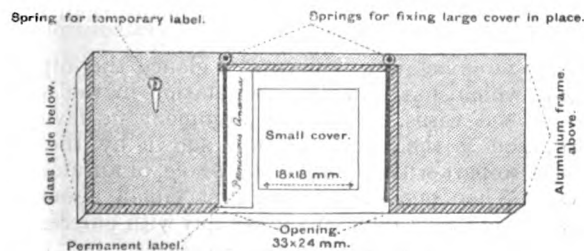
As regards *treatment*, I afterwards regretted that in both Cases I. and II. I had not at once incised the places bitten and dissected out the damaged subcutaneous tissue immediately beneath the fang marks, as I would have done had I seen the cases early. Even in cases that come late for treatment it is well to remove this damaged tissue, in which presumably any of the poison that remains still unabsorbed will be lying. Permanganate of potash may then be rubbed into the wound.

CAN WE DO WITHOUT SLIDES?

By EDWARD HORDER, F.R.C.S. Edin.

MESSRS. ZEISS, of Jena, and 89, Margaret Street, London, have made for me an aluminum frame, the object of which is to obviate the necessity of carrying a large number of the usual 3 × 1 in. glass slides.

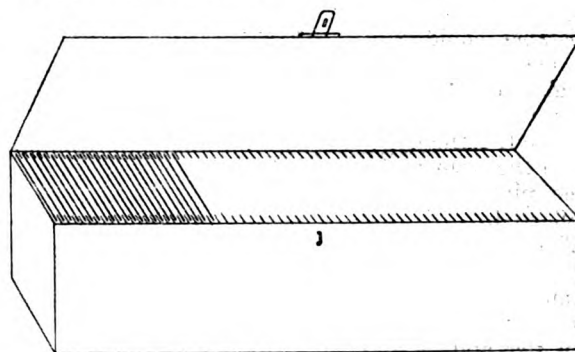
The frame is 76 × 26 mm. (3 × 1 in.), with an opening of 33 × 24 mm. A glass slide, 76 × 26 mm. is attached to the under surface of the frame by means of four plated screws. There are two springs in the opening for holding a large cover-glass, 32 × 24 mm., firmly to the glass slide, and also a small spring on the frame for securing a provisional label.



To prepare the frame for use, a 32 × 24 mm. cover, which can be purchased from Zeiss or elsewhere, is laid on the glass slide beneath the two springs; the

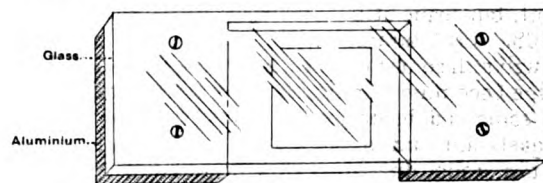
specimen, on a smaller cover, is placed on the centre of the larger one, and is now ready for examination. For specimens which are to be stained the blood can be spread on either cover, but for wet ones, requiring immediate examination, the smaller cover should receive the drop of blood.

The glass slide beneath the frame is made of the best white plate glass and will, with ordinary care, need but an occasional renewal. Messrs. Zeiss supply extra glass slides with each frame. The two sizes of covers, 32 × 24 mm. and 18 × 18 mm., with the frame, are the only apparatus required for all ordinary clinical work, the frame rendering the carrying of slides quite unnecessary.

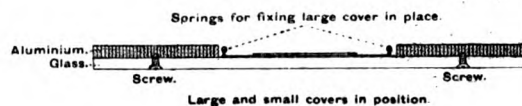


Box for carrying covers.

It is believed that this frame will be a help to many, especially to those who are obliged, either from choice or duty, to travel shorter or longer distances, and with whom every ounce is a consideration, not to mention the deterioration of glass slides. To such, a packet of covers will be all that is required, the staining and examination by means of frame being postponed until their return.



Lower surface—showing (1) glass slide with aluminium frame attached, (2) opening through the glass, and (3) four plated screws, by which frame is fastened to screw.



Section through "opening."

Another advantage is that the storing or preservation of specimens requires but an infinitesimal amount of space compared with the ordinary 3 × 1 in. slides; and the great difference of weight is also in favour of covers and frame. One hundred specimens, when covers are used, can be packed away in a box 6 × 1½ × 1½ ins. large.

This box, made of aluminium, is remarkably light. The sketch shows projecting teeth, between which the specimens are placed end-ways. But any box, one

half the size, will be found large enough for carrying the same number of specimens safely, if separated from each other by means of their drying paper. On the larger of the two covers sufficient space will be found for a permanent label.

The frame and box being made of metal no danger of warping or breakage need be feared, two strong recommendations to those working in the tropics.

As the frame bears the name of Zeiss, it may be scarcely necessary for me to add it is both beautifully and perfectly finished.

I should mention that the dimensions of the "opening" can be made to fit any cover-glass, provided the size required be given at time of ordering.

AMPUTATION FOR PERFORATING ULCER OF FOOT IN LEPERS.

By EDWARD HORDER, F.R.C.S.Edin.

Physician Superintendent, Leper Asylum (140 beds) and General Mission Hospital, Church Missionary Society, Pakhot, China.

ABOUT two years ago I wrote Dr. Manson that, arguing from tuberculosis, in which disease every attempt is made to remove sources of sepsis, amputation for perforating ulcers of the feet of lepers should give good results, and therefore be performed, in order to remove once for all the source of auto-infection, eradicate as far as possible the cause of the exacerbations of leprosy, and give these patients another chance of obtaining fairly good health.

Dr. Manson replied that he thought judicious amputation under such circumstances to be good surgery, and kindly mentioned in his second edition of "Tropical Diseases" that I "strongly recommended amputation for perforating and other forms of ulceration; the general health is much improved by the removal of such sources of sepsis" (see article on Leprosy, page 453).

More than two years have passed since the first operation was performed, and knowing it has been stated that amputation of an ulcerated foot of a leper is useless, because the stump is sure to break down, I now send, to show this is not always the case, particulars of nine consecutive cases, which were operated upon by my colleague, Dr. L. G. Hill. Less than amputation is of no avail; removal of necrosed bones and tendons has not given permanent relief in our hands.

Case 1.—Female leper, Chan I., aged 18. Foot had been in an ulcerated condition for four years. Patient had suffered much from outbreaks of fever all the time, and never felt well. The right foot was removed by Syme's operation on October 23rd, 1899. Patient made a good recovery and is much stronger. There has been *no ulceration* since operation.

Case 2.—Female leper, Ne I., aged 16. Foot with perforating ulcer for four years, and constantly suffering from ill-health and fever. The left foot was removed on October 23rd, 1899, by Lisfranc's operation, and for eighteen months the stump was in an excellent condition. During the last nine months there has been *slight ulcerations*, but no fever since the operation. Patient feels much stronger.

Case 3.—Male leper, Yeung Tsat, aged 25. Foot ulcerated for many years, and patient suffering from constant outbreaks of leprosy and fever. A Lisfranc was performed on October 9th, 1899, and there has *not been the slightest ulceration* since, neither has the patient suffered from any exacerbations of leprosy.

Case 4.—Male leper, Lan Lok, aged 39. Very badly ulcerated foot, patient refused amputation for a long time. A Lisfranc was performed on August 2nd, 1899, but within a few months the stump broke down, and remained in this condition all the time the patient resided in the Asylum. Amputation of the leg would have given better results.

Case 5.—Male leper, Che Sam, aged 26. Perforating ulcer was present in the right foot and he suffered much in general health. A Syme was performed on August 5th, 1899, and the patient has remained in excellent health. *No ulceration* since operation.

Case 6.—Male, leper, Po Lo, aged 23. Suffered much from a perforating ulcer of foot, also fever and exacerbations of leprosy for about two years. Often on the sick list. Foot was removed by Lisfranc's operation on August 10th, 1899. Patient is much stronger, and foot has *not ulcerated* since operation.

Case 7.—Male leper, Ho Shan, aged 27. Badly ulcerated right foot for eight years, exacerbations and fever every few months, when foot and leg swelled. Patient never felt well. Foot removed by Parabeuf's subastragaloid amputation on October 4th, 1899. Patient has felt much stronger, and the stump has *not ulcerated*. During the last year he has taken salicylate of soda, and states "he never felt better."

Case 8.—Male leper, Sz Tai Fan, aged 28. Complained much of fever, weakness and general bad health. Left foot ulcerated for eleven years. This was amputated on October 9th, 1899, by Lisfranc's operation, but has not been a great success. There was *no ulceration for a year*, but since then small areas have broken down. These heal, but give way in a few months, to heal again. The patient, however, asserts he is much stronger than before the operation, and rarely suffers from fever.

Case 9.—Male leper, Lam Han Yeung, aged 25. Left foot with deep perforating ulcer for four years. Amputated on August 9th, 1899, by Lisfranc's operation. Patient states that before the operation he had exacerbations of leprosy about twice each year, when the foot swelled and became very painful. For one year after the operation there was no ulceration of the stump, but since then a portion was broken down. He is in a much better condition of health than before the removal of the foot.

The table below will show at a glance the improvement which has followed amputation in the above-mentioned cases. A strong argument in favour of operation is the readiness now shown by the poor lepers to part with a portion, or whole, of an ulcerated foot. When operations were first talked about, the lepers were very reluctant to comply with our request, much persuasion often being necessary; but to-day they will ask for an amputation, knowing better health for a long period is insured for them by the removal of the ulcerated foot. In the above notes "fever" and "exacerbations of leprosy" signify the

effects seen in a patient after a fresh dose of toxin has been absorbed, either from an ulcerated foot or internal organs. At such times the leprosy bacillus can usually be found in the blood. There is a fresh febrile attack consequent on a new liberation of bacilli and their toxins. The removal of the cause where possible prevents this auto-infection and saves the patient from these recurring outbreaks.

Patients in the leper asylum are prescribed chaulmoogra oil, gurgun oil, and cod liver oil, also arsenic and acids. A few cases have been taking salicylates with good results. Many drugs with an advertised "cure for leprosy" have been given from time to time, only to make us more thankful for the old remedies just mentioned. It is the concomitant diseases from which the lepers suffer that require so much time and attention.

It is very noticeable how very limited is the life of many of the lepers after they leave the asylum, proving that good housing needed for ordinary complaints are factors to be reckoned with in the treatment of this dire disease.

| Name | Sex | Date of Amputation | Operation | General Health | Return of Ulceration | Return of Exacerbation | No. of years foot ulcerated |
|---------------|-----|--------------------|-----------|----------------|-------------------------------------|------------------------|-----------------------------|
| Chan I .. | F. | 1899. Oct. 23 | Syme | Much improved | None .. | None | 4 |
| Ne I .. | F. | " " | Lisfranc | " " | Not for eighteen months | " | 4 |
| Yeung Tsat | M. | " 9 | " | " " | None .. | " | Many years |
| Lan Lok | M. | Aug. 2 | " | Not improved | Yes .. | Yes | " |
| Che Sam | M. | " 5 | Syme | Much improved | None .. | None | (?) |
| Po Lo .. | M. | " 10 | Lisfranc | " " | None .. | " | 2 |
| Ho San .. | M. | Oct. 4 | Fara-beuf | " " | None .. | " | 8 |
| Sz Tai Fan | M. | " 9 | Lisfranc | Improved | Not for a year | Rarely | 11 |
| Lam Han Yeung | M. | Aug. 9 | " | Improved | Not for a year, slightly at present | Rarely | 4 |

BERI-BERI.

At the last meeting of the Epidemiological Society, Dr. Patrick Manson entered very fully into the question of the etiology of beri-beri, and although it is unfortunately true that this question still remains unsolved, yet there was much in what he said which was very suggestive, not only in relation to beri-beri itself, but also in regard to the causation of various other diseases. To those of us who practise in this country beri-beri may perhaps appear a matter of small interest, but the extraordinary gravity of the disease in regions where it prevails may be judged of from the statement made by Dr. Manson that in some plantations over 75 per cent. of the coolies have been killed off by beri-beri in a single year. Two theories are at present held as to the etiology of the disease, viz., the dietetic and the microbic. Among

those who have urged the dietetic theory, those have seemed to have most reason on their side who have attributed the disease to prolonged nitrogen starvation, owing to the long-continued use of a uniform rice diet. In proof of this theory some very striking cases have been collected in which after an improvement has been made in the diet the disease, previously very prevalent, has rapidly disappeared. On the other hand, this theory fails to explain all the facts, for numerous examples can be brought forward which show that the disease may prevail among populations whose diet has been specially devised so as to avoid the defects to which the origin of beri-beri has been attributed, and there are also many instances on record of Europeans enjoying a liberal diet falling victims to the disease. As to the germ theory, Dr. Manson, although saying that it is the more plausible of the two, adds that whether the germ produces its morbid effects while proliferating in the human body, or whether it acts indirectly by producing outside the body a toxin which, on being ingested or otherwise absorbed, acts on the nerves, it is impossible to say. On the whole, Dr. Manson inclines to the latter hypothesis, namely, that beri-beri is purely an intoxication, produced by a toxin elaborated by a germ whose nidus is located outside the human body, and "that in this respect beri-beri is on all fours with alcoholism, the germ of which is the yeast plant, the nidus solutions of sugar, the toxin alcohol, and, to complete the parallel, the pathological effect, a peripheral neuritis." That the disease is due to a living germ is proved by the facts that (a) the cause can be transported from place to place, and therefore cannot be of a climatic or meteorological nature; and (b) that when so transported it can multiply and spread, and therefore cannot be of an inorganic nature. The great question is where does the germ live and grow and produce the toxin by which the disease is caused? In answer to this it can at least be shown that certain ships become infected with beri-beri, and if ships why not houses and localities? There is a marked tendency among those who have studied this subject to regard rice as at least the favourite nidus of the disease, but, at any rate, whatever the exact medium in which the poison is distilled, the malady may be regarded as a "place disease," and it is much to be hoped that the expedition which has gone out to study its etiology will be able to isolate the organism by which it is caused and to discover the favourite media in which it grows, and by aid of which it becomes endemic in certain localities. We need hardly point out what an important subject is opened up for inquiry by the suggestive remarks of Dr. Manson. Who is to say what maladies and what conditions of deteriorated health may not arise from the absorption by the human body of the toxins produced by micro-organisms which have their *locale* in the food, the drink, the raiment, and the houses of mankind? We already know something of ptomaine poisoning, of alcoholism, of pellagra, of ergotism. Now we are told about beri-beri, but who shall say where is the end of the list of diseases which may be called zymotic in that they are due to "germs," but the germs of which are extra corporeal micro-organisms?—*The Hospital*, November 30th, 1901.

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THE

Journal of Tropical Medicine

JANUARY 1, 1902.

THE FUTURE OF THE JOURNAL.

It is now twelve months since we ventured upon the publication of this Journal twice monthly. We did so at the suggestion of several contributors and subscribers, and we have no reason to regret the step. We have received many congratulatory letters on the subject, and, as the list of subscribers gradually extends, we believe the Journal is fulfilling, however imperfectly, the objects for which it was started. We are thankful for expressions of opinion: naturally we welcome those of a laudatory nature, but no less do we appreciate criticisms pointing out where we fail and where we might improve.

We urge upon our supporters to write to us *direct* when they find causes for complaint; they will be listened to in an appreciative spirit, for the Journal exists for the subscribers and not

for its editors and publishers. It is intended to be a direct vehicle of communication between medical men in distant lands; to be a means of voicing their wants and requirements; and to bring about a community of interest amongst the members of the medical profession who have to deal with diseases other than those met with, in the days of their professional education, in the medical schools of these islands.

We have been urged by some to form a "Tropical Medical Association," by others a "Colonial Medical Association," and to make the JOURNAL OF TROPICAL MEDICINE the official organ of such a society. We are willing to consider the formation of such a society seriously, if in the opinion of the supporters of the Journal it is thought desirable. It would not imply any extra subscription, but it would serve as a closer bond of union between medical men in countries far apart, but united by the fact that their surroundings and daily professional life are in spheres which have much in common.

Were such a society formed we would be enabled to have a "Journal Committee," to which the communications, &c., to the Journal would be submitted, and the opinion of the Committee would necessarily dominate the spirit and letter of the publications. It has even been suggested that the Journal might be formed into a public company, and that those interested in the subject of tropical medicine might become shareholders in the Journal, and directly control not only its literary but all its financial affairs. Whether this is likely to prove attractive, to the more commercially inclined, it is not for us to say, but an expression of opinion on the subject would have, no doubt, influence with the proprietor.

It is not usual for scientific journals to discuss matters of this kind in their columns, but the Editors feel that the Journal is a publication of so special a kind, appeals directly to so large a number of medical men who are specialists in tropical diseases, and is so important an element in the question of the hygiene of the Empire, that it ought to be established on the broadest possible basis.

THE ADMINISTRATION OF QUININE IN LAGOS.

H. E. SIR WM. MACGREGOR, G.C.M.G., LL.D., M.D., Governor of Lagos, sends the following communication regarding the administration of quinine in Lagos.

His Excellency remarks that the lists will show that the omission to take quinine runs in parallel lines with ignorance. The educated men who write grammatically take quinine, the others do not. Of the 174 who did not take quinine the answers of two groups of twenty officers, namely, the first twenty and last twenty, are taken as examples. That Sir Wm. MacGregor's remarks are amply justified may be gathered from the perusal of the illiterate returns and the nature of the reasons assigned for not taking quinine.

RETURN OF OFFICERS WHO DO NOT TAKE QUININE AT ALL, AND REASONS ASSIGNED FOR NOT TAKING THIS DRUG.

| No. | Reasons. |
|-----------|--|
| 1 | I have never taken Quinine; and in fact do not know what it is. |
| 2 | No cause. |
| 3 | I scarcely have an attack of Fever and whenever I do, I use the <i>Agunmu</i> . |
| 4 | Native Medicine is more preferable. |
| 5 | Native Medicine is more preferable. |
| 6 | As I use Native Remedies for fever. |
| 7 | For I have no cause for it. |
| 8 | Because I have not the cause of taking it. |
| 9 | I am not accustomed to English Medicines. |
| 10 | I don't know how to take it. |
| 11 | I have never taken it, as it has never been given me by any medical Officer. |
| 12 | I got no Fever. |
| 13 | Because I have nobody to supply me. |
| 14 | Simply because there is no one to supply me. |
| 15 | None at hand presently. |
| 16 | Native Medicine is more preferable. |
| 17 | Because am not supplied. |
| 18 | I see no necessity for taking quinine as a preventive since I seldom suffer from fever. |
| 19 | Native Medicine is more preferable. |
| 20 | Because I have not been accustomed to do so. |
| * * * * * | |
| 155 | Because I was not trained up with it. |
| 156 | I never accustomed to such drug. |
| 157 | Is not agreeable with me. |
| 158 | Because it is not used to. |
| 159 | Because there is no occasion for it at present. |
| 160 | I use native medicine. |
| 161 | Because I use the Native medicine for fever. |
| 162 | My reason for not taken quinine is that I have never taken any before and sometimes when feverish I take Fruit Salt having no idea of the quinine as preventive. |
| 163 | Because I had not once suffered from Fever. |
| 164 | I never taken such medicine because I am accustomed with native medicine. |
| 165 | Because I have not come across it before. |
| 166 | No necessity needs this. |
| 167 | Because I never suffer from Fever. |

- 168 Because I never use any before, I always use Native Medicine.
- 169 Because I do not know that it is a preventive.
- 170 I take no quinine because I have had no Fever ever since I have been to this District, and I am not in supply to use it as a preventive.
- 171 Because I am accustomed to Native Medicine.
- 172 Because native treatment has been found agreeable with me.
- 173 Simply on account that I am accustomed to Native Medicine.
- 174 Because I had it taken sometimes ago by the Doctor I felt something very bad in my head and for couple days after I hardly could hear when speaking.

RETURN OF OFFICERS WHO TAKE QUININE REGULARLY.

Seventy-six officers took quinine as a prophylactic and for the most part in the following doses:—

| | Quantity and time. | No. |
|----------|--|-----|
| 2 | grains daily | 1 |
| 2½ | " " | 3 |
| 3 | " " | 3 |
| 4 | " " | 1 |
| 5 | " " | 48 |
| 10 | " " | 1 |
| 2 | " weekly | 1 |
| 8 | " " | 2 |
| 10 | " " | 2 |
| 15 | " " | 2 |
| 10 to 15 | " " | 2 |
| 10 to 50 | " " | 1 |
| 5 | " twice weekly | 1 |
| 15 | " occasionally for 3 consecutive days | 1 |
| 15 | " for 2 consecutive days repeated after 8 days | 1 |
| 5 | " every other day | 3 |
| 10 | " " " | 1 |
| 4 | " twice monthly | 1 |
| 10 | " monthly | 1 |
| | | 76 |

LEPERS IN ENGLAND.—In the recently issued report of the medical officer for the Port of London, Dr. Collingridge describes how he received information from the owners of the sailing ship *Fingal* that one of the crew of that vessel shipped at Calcutta, was found to be a leper, and that they had been forbidden by the United States Government to land the man at San Francisco. On the vessel's arrival at Gravesend "the patient was removed to the West London Hospital at the expense of the owners, this disease not being one dealt with under the Public Health Act." It is not to be expected, however, that the owners will make themselves permanently chargeable with this patient, so here we have another "undesirable" dumped upon our shores. Whether or not, coming from Calcutta, he is a fellow subject, or merely a casual picked up at that cosmopolitan port of call, we do not know. The point is that while other countries refuse to receive these people we admit them without question.

A MONOGRAPH
OF THE
CULICIDÆ
OR
MOSQUITOES.
MAINLY COMPILED FROM THE COLLECTIONS
RECEIVED AT
THE BRITISH MUSEUM
FROM VARIOUS PARTS OF THE WORLD
IN CONNECTION WITH
THE INVESTIGATION INTO THE CAUSE OF MALARIA
CONDUCTED BY THE COLONIAL OFFICE
AND THE ROYAL SOCIETY.

BY
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*Author of "A Text-Book of Agricultural Zoology,"
"The Parasitic Diseases of Poultry," etc.*

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1901.

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PRELIMINARY NOTICE.

THE notice on the adjacent column is not an advertisement, and we have departed from the usual stereotyped form of drawing attention to this work on account of its scientific and national importance.

The rôle played by mosquitoes in the spread of malaria, filaria, and probably yellow fever, &c., is so definite, so circumstantial, and so pregnant with possibilities in practical prophylaxis that too much attention cannot be bestowed upon the zoology of these insects. It is impossible to enter into detailed criticism of a work of such primary importance, seeing that the monograph has been but a few hours in our hands; yet a glance at the three handsome volumes justifies the conclusion that we have before us a scientific work which will rank as a classic.

Volume I. extends to 424 pages, and is illustrated by no less than 151 accurate drawings. Volume II consists of 391 pages with 166 illustrations. Volume III. is devoted to plates, of which 148 are coloured. The work is produced in a style creditable alike to the author, the artists, the printers and publishers. The scientific accuracy of the drawings and colourings may be relied upon, for they have been produced with infinite care and with a scientific accuracy of which it is impossible to speak too highly.

Mr. Fred. V. Theobald is so well known as an authority, perhaps the highest authority living, on the subject of the *Culicidæ*, that we accept his descriptions with the knowledge that we have before us a standard of reference in the work he has produced. The first paragraph of the preface contains a statement highly creditable to the Secretary of State for the Colonies, to whom practical science, especially in the field of tropical medicine, is already so deeply indebted. Professor E. Ray Lankester, the Director of the Natural History Department of the British Museum, who writes the preface, states, "The present 'Monograph of the Culicidæ or Mosquitoes,' had its origin in the appointment by the Royal Society, at the request of the Right Hon. Joseph Chamberlain, of a committee to co-operate with the officials of the Colonial Office in the investigation of the causes of malaria and the possibility of controlling that scourge of tropical lands."

Professor Lankester "sought the assistance of the Colonial Office, the Foreign Office, and the India Office in the attempt to procure properly preserved collections of mosquitoes from all parts of the world."

We are so accustomed in Britain to find all scientific investigation left to private and individual enterprise, that the present departure by those great departments is a welcome factor in our national life, and an indication that the physical welfare of peoples of the Empire, which has been so long officially neglected, is at last receiving the attention it deserves.

We give the addresses where this important work may be obtained advisedly, for we are of opinion that it is as important for practitioners in warm climates to have this monograph in their hands as to have the most recent work on general medicine.

News and Notes.

At the London School of Tropical Medicine, during the session just finished, twenty-eight students attended the course. Several applicants were refused owing to want of laboratory accommodation. The Seamen's Hospital Society and the School authorities are engaged in planning an extension of the School premises.

MR. ALFRED JONES, the originator of the Liverpool School of Tropical Medicine, has been created a Knight Commander of the Order of St. Michael and St. George. In connection with the School, and largely owing to the initiative of Sir Alfred Jones, a hall of residence for students has been arranged in Liverpool within easy residence of the Medical School and the Royal Southern Hospital. The cost of residence is wonderfully moderate, and it is hoped to attract students from the tropics, the hall being open to all races and creeds.

IRRIGATION IN INDIA.—It is a pathetic spectacle, that of Major-General G. C. Cotton, C.S.I., in the ninety-fourth year of his age vigorously carrying on the evangel of irrigation in India, which his brother, the famous Madras engineer, Sir Arthur Cotton, started so long ago as 1834. From his retirement the earnest nonagenarian follows with close interest the course of events in this country, and loses no opportunity of bringing the object which is nearest his heart prominently before the public. He has just issued, through the well-known publishing house of Rivingtons, a remarkable pamphlet containing a letter and two other papers on the waters of the great rivers of India as essential to the nation and the only possible means of preventing famine. The pamphlet well deserves the serious study of all interested in the important subject of irrigation in India. — *Indian Engineering*, September 21st, 1901.

CHILDREN KILLED BY ANTITOXIN.—A great sensation has been caused in St. Louis, U.S.A., by the announcement that eleven children have just died as the result of antitoxin administered to them as part of treatment for diphtheria. The eleventh child died on October 31st, and there were twenty-five other sufferers. The cause of death in each of the eleven cases was tetanus, and the physicians in attendance are unanimously of opinion that it was brought about by the antitoxin.—*Brit. and Col. Druggist*, November 8th, 1901.

SANITATION OF CUBA.—A Cuban physician, Dr. A. Bustillo Lirola, writes in the *Annals of Gynecology and Pediatrics* on this subject. He compares the conditions prevailing in the island under Spanish rule and under American, needless to say, greatly in favour of the latter. He says: "We can proudly say that Cuba is hygienised. This could be possible, because the true Cuban population have aided greatly in it. Yellow fever has diminished in such a notable way that this fact alone is noteworthy. During last June

there has not been a death from yellow fever, nor even a case of the disease. Records show that since 1781, no previous June has passed with absolute freedom from the disease. Infectious diseases, as a rule, are less frequent. With the war on mosquitoes, malarial fever has also diminished. Leaving out of consideration the death-rate from 1895-1898, if the number of deaths during the year 1900 is compared with that of the years previous to 1895, we shall find that they are much less now. This is valuable testimony to the efficient efforts made under American occupation to render Cuba healthy, the more so, coming, as it does, from an impartial source."

A WOMEN'S HOSPITAL AT MANILA has been opened recently, and is ready for the reception of patients. Miss Mary MacDonald, a graduate of Bellevue Hospital, who had a wide experience in military hospitals during the Spanish-American war, and has since seen conspicuous service in Manila, is the superintendent.

The founding of the hospital, which was a much-needed institution, was largely due to the generosity of Mrs. Whitelaw Reid, who expressed her willingness to give 5,000 dollars toward the founding of such an institution. A board of trustees was immediately named, consisting of prominent Manila officials and business men, and the work was put into immediate operation.

The present service is limited to fifteen beds, but on occasion increased accommodations could readily be obtained. The furnishing of the rooms and all the accessories are of the latest and most approved pattern. The operating room, which is thoroughly modern, was equipped through the generosity of Colonel Greenleaf, Surgeon-General to the American army in the Philippines.

THE SIAMESE popular system of medicine is very much like the Chinese in many ways. Here is a recipe for a cold: "One portion of rhinoceros horn, one of elephant's tusk, one of tiger's, and the same of crocodile's teeth, one of bear's tooth, one portion composed of three-parts bones of vulture, raven and goose, one portion of bison, and another of stag's horn, and one portion of sandal. These ingredients to be mixed together on a stone with pure water. One-half of the mixture to be swallowed, the rest to be rubbed into the body."

THE LATE SURGEON-GENERAL HARVEY. GOVERNMENT RESOLUTION.—The following appears in the *Gazette of India*: "The Governor-General in Council has received with much regret the intelligence of the death at Simla on the 1st instant of Surgeon-General Harvey, Director-General, Indian Medical Service.

"Surgeon-General Harvey had only just returned from furlough to resume the duties of the Director-Generalship, a post to which he was first appointed in February, 1898, after honourable service extending over more than thirty years. By his death towards the close of a long and distinguished career the Government lost a valued public servant and trusted adviser."

DR. THORBURN MANSON leaves for Christmas Islands on Friday, January 3rd, to take up his duties as one of the members of the Commission recently despatched thither to investigate and report upon beri-beri.

PLAGUE.

PREVALENCE OF THE DISEASE.

At the present moment, January 1st, 1902, plague exists as a scourge only in India and in Mauritius.

INDIA.—During the weeks ending November 30th and December 7th the number of plague deaths throughout all India numbered 7,961 and 8,878 respectively.

In the Bombay Presidency plague is widely distributed, 5,655 and 5,989 fatal cases (out of the total for all India) having occurred in this Presidency alone during the last week of November and the first week of December.

MAURITIUS.—For the week ending December 26th 38 cases of plague and 23 deaths from the disease occurred in Mauritius.

EGYPT.—Since November 29th no death from plague has been reported in Egypt. Only one case of plague was reported during the week ending December 15th in Egypt.

CAPE OF GOOD HOPE.—During the second week of December, 2 cases of plague only were reported from Cape Colony; one patient resided at Mossel Bay, the other at Ladismith. During the third week of December two natives developed plague at Port Elizabeth, but there were no cases elsewhere; no death from plague occurred during the week.

Current Literature.

DIARRHŒA AND DYSENTERY.

THE ACTION OF TANNIGEN IN DYSENTERIFORM ENTERITIS.—Schweiger, who has had a considerable experience in Bosnia of the treatment of enteritis, complicated in the majority of cases by hæmorrhage (this form is known as "Bosnian disease"), bears testimony to the favourable action of tannigen. In twenty cases of hæmorrhagic enterocolitis tannigen was administered; usually opiates are given, with the serious drawback that meteorism is often produced. If, on the contrary, tannigen is prescribed ($7\frac{1}{2}$ grains three times a day), this symptom is not met with. All the cases treated with tannigen were easily cured without recourse to any other drug.

In chronic dysentery the results are less brilliant, but large doses (15 grains three times a day) effect considerable improvement.—F. H. S., *Die Heilkunde*, July, 1901.

SULPHUR IN DYSENTERY.—The treatment of dysentery was summarised by Sir William Gull in three words—"rest, warmth, and ipecacuanha."

However, in many cases these measures prove disappointing. Remembering that sulphur is an efficient substitute for ipecac. in the local treatment of anthrax, Dr. G. E. Richmond decided to try the same substitute in dysentery. He prescribed 20 grains of sublimed sulphur with 5 grains of Dover's powder every four hours. The two cases in which the treatment was carried out both bear testimony to the value of sulphur in this affection. It is no specific, but a welcome addition to our remedies. As soon as the diarrhœa diminishes, less frequent doses should be given, otherwise obstinate constipation will follow. The remedy seems to exert an antiseptic action.

Pain and tenesmus are relieved by sulphur more effectually than under other methods of treatment. Moreover, the cure obtained seems to be more lasting, the disease showing less tendency to become chronic or to relapse. Bloody and mucous stools rapidly give place to fæcal evacuations, and the odour of the fæces becomes less offensive, though the number of movements does not at once diminish.

The author has not met with a case of dysentery which would not yield to the sulphur treatment.—*Medical Record*, October 19th, 1901.

RHABDONEMA INTESTINALE AND INTESTINAL FLUX.—The exact part played in the diarrhœas and dysenteries of hot climates by the *Rhabdonema intestinale*, or *anguillula stercoralis*, as its better-known rhabditiform phase is called, has always been a matter of uncertainty since the nematode was discovered by Normand in 1876. It is generally believed to be harmless; Manson thinks it doubtful if it can even produce a slight degree of intestinal catarrh, and Brault, "Maladies des Pays Chauds," p. 107, sums up thus a discussion on the worm, "it exists simply as a sort of epiphenomenon, the anguillula is introduced accidentally into the digestive system, because it finds in the condition of the intestinal secretions a milieu favourable to its existence and reproduction." More recently, however, Dr. Strong, Director of the Army Pathological Laboratory at Manila, reports on a case of a patient, eight years a resident of Baltimore, U.S.A. (*Johns Hopkins Bulletin*, x., Nos. 1, 2, 1901), who suffered from chronic diarrhœa and liver abscesses; in the pus of the abscesses amœbæ were found, and a microscopic examination of the stools showed embryos of the anguillula and amœbæ. At the autopsy the "parasitic form" of the adult female *Rhabdonema* was found in the small intestine. Strong refers to four other cases of infection by this worm, causing intermittent diarrhœa and gastrointestinal disturbances. In the Baltimore case "the eggs, embryos and the worms" were found in the glands of Lieberkühn, where they produced atrophy of the epithelium and round-celled infiltration.—*Philadelphia Medical Journal*, August 31st, 1901.

GOUDOU OR ANAKHRE.—Drs. Mendes and Jeanselme describe this disease, which is prevalent amongst the negroes of the West African coast, as a "bony, spongy tumour, ovoid and symmetrical, growing at the root of the nose." The disease as a rule sets in before puberty, and by its encroachment on the eye

and nose it interferes with the sight and hampers breathing. The general health remains comparatively unimpaired, but headache, a muco-purulent discharge from the nose, and epistaxis herald the appearance of the ailment, Mendes observed a case in Brazil; this had the peculiarity of being unilateral. Extirpation of the growth is the only effective remedy of the disease.—*Revue de Chirurgie*, October, 1901.

FORMULÆ.

THE ADMINISTRATION OF QUININE TO CHILDREN.—The following prescriptions are cited by the *Revue médicale* for October 9th from an analytical article by Dr. Lemanski in the *Bulletin de l'hôpital du Tunis*.

Petzold gives quinine in honey dissolved in acidulated water:

R Quinine sulphate 10 grs.
Acidulated water 75 mins.
White honey 600 grs.

M.—A coffeespoonful every two or three hours.

Crépu's prescription:—

R Distilled water 600 mins.
Extract of liquorice 45 grs.
Quinine hydrochloride 4½ grs.

M.

A child usually makes no difficulty in swallowing the whole of this at one time.

A recent number of the *Klinische therapeutische Wochenschrift* gave the following:—

R Quinine sulphate 60 grs.
Citric acid }
Syrup } of each, 150 grs.
Syrup of bitter orange peel }
Distilled water 300 mins.

M.

Ten drops of this mixture are given in an ounce and a half of water, to which are added forty-five grains of sodium bicarbonate. The mixture is drunk while effervescing.

Saccharine may also be used advantageously to mask the taste of quinine.

Dr. Lemanski, himself, prefers the rectal method of administration. The suppository is better tolerated than the enema and causes no smarting or defæcation.

R Quinine salt from 1½ to 7½ grs.
(according to age)
Cacao butter from 15 to 45 grs.
Virgin wax q. s.

M. ft. suppositorium.

In some cases cacao butter may be replaced by glycerin solidified by the addition of gelatin. Two suppositories daily are to be inserted without regard to the thermometrical exacerbation, the object being to saturate the economy with the drug for a sufficiently long time to combat the paludism.—*N.Y. Med. Journal*, November 16, 1901.

FOR MALARIAL LIVER TROUBLE.—M. Lemanski (*Nouveaux Remèdes*, October 8th) gives the following:—

R Sodium cacodylate ⅓ of a gr.
Powdered cinchona }
Sodium bicarbonate } of each 7½ grs.

M.

For one wafer. Three wafers to be taken daily.

Frequent purgation, by means of the following formula, is also often effective:—

R Calomel 12 gra.
Cascara }
Powdered rhubarb } of each 9 ,,
Powdered belladonna 7½ ,,

M.

Divide among three wafers, to be taken fasting in the morning, at intervals of a quarter of an hour, once a week.

TREATMENT OF DYSENTERY.—Hughes recommends the following in the treatment of dysentery:—

R Acidi sulphurici dil 3ss
Tinct. opii deod. 3i
Spts. camphoræ, aa 3i
Tinct. capsici 3ss
Spts. chloroformi, aa 3ss
Spts. vini gallici 3iss
M. Sig.—One teaspoonful every four hours; or:—
R Magnesii sulphatis 3iss
Acidi sulph. dil. 3ii
Tinct. opii deodor., aa 3ii
Aq. chloroformi q. s. ad. .. 3iii

M. Sig.—One dessertspoonful every four hours.

—*Med. Record*.

LEPROSY.

DR. HALLOPEAU in an article in *Lepre*, June, 1901, advocates the use of chaulmoogra oil subcutaneously in cases of leprosy in which it causes digestive disorders when taken orally. In confirmation of these views he quotes a case of Tourtoulis-Bey of Cairo:—

"In this case the subcutaneous injections were made at first very frequently, about twenty times a month, 5 gm. at a time, and were continued at intervals for about five years. The injections were made on the extensor surfaces of the extremities, were not painful, and the swelling that they caused disappeared in twenty-four hours. The improvement began at once, until finally the patient could be regarded as cured, having only a few slight signs of the trouble left."

Hallopeau has come to the conclusion that:—

(1) Lepers treated with chaulmoogra oil, either hypodermically or by the mouth, may show such an improvement that they may be fairly regarded as cured.

(2) More often they continue to show signs of the affection, but these are usually of a benign character.

(3) In certain cases intense local and general exacerbations appear in spite of the treatment.

(4) It is probable that this drug has a favourable influence upon leprosy.

(5) This influence is not usually sufficient to cure the disease; it cannot in any way be compared to that of mercury or the iodide of potash in syphilis, nor to that of quinine in malaria.

(6) It is best given in hypodermic injections to patients who bear it well; it may give rise to febrile disturbances and to pulmonary emboli.

MALARIA AND MOSQUITOES.

FORMALDEHYDE GAS FOR DESTROYING MOSQUITOES.—Dr. M. J. Rosenau declares, in a recent issue of *Public Health Reports*, that experiments undertaken by him have shown that formaldehyde gas is an efficient

insecticide so far as the mosquito is concerned. The experiments were all made upon the *Culex pungens*, and it was found that an exposure of three hours was invariably sufficient to kill all the mosquitoes of this variety in a confined space, provided the gas was used in amounts usually employed for disinfection.

MOSQUITOES ATTRACTED BY SOUND.—Major Ronald Ross writes to the *British Medical Journal* that he has recently received a communication from Mr. Brennan, of the Public Works Department, Jamaica, containing the following observation: "You will pardon me for drawing your attention to the fact, if you have not already noticed it, that the mosquitoes (I do not know if every variety) will respond to such sounds as a continuous whoop or hum. I have tried the experience lately, and find swarms gather round my head when I make a continuous whoop. There may be, however, some particular note or pitch that would be more attractive to them." This would afford an interesting subject for investigation, the journal quoted remarks, on the lines of Dr. Nuttall's recent research on the colours attractive to mosquitoes.

MALARIAL HÆMOGLOBINURIA.—Otto Lerch says that in the treatment of this condition, it must be borne in mind that quinine has no curative influence; it destroys the plasmodia, but if enough has been taken to accomplish this object, we aggravate the case by adding one more toxic substance to those already present. Absolute rest is of paramount importance. Fresh air is the next indication. The primary lesion is an intense anæmia; the red blood-corpuscles are enormously decreased in number, their chemical composition is changed, and they are especially deficient in hæmoglobin, so that the physician should see to it that every corpuscle that is able to carry oxygen should have a chance to grasp it. Pure air is the best thing, but inhalations of oxygen may also be tried. The emunctories must be cleared, the anæmia is to be treated with iron and arsenic, and the diet must be carefully regulated.—*New Orleans Medical and Surgical Journal*, November, 1901.

PSYCHICAL DISTURBANCES IN PALUDISM.—M. Jean P. Cardamatis says that one must distinguish between the psychical disturbances which occur in the height of a simple intermittent fever, those which arise during the fever and in the intermittent stages of chronic paludism, those observed in the course of a remittent fever, and the psychoses which appear in the course of malarial cachexia. These manifestations are undoubtedly due to the toxins developed during the disease, and usually in persons of a neuropathic taint. Paludism can arouse morbid predispositions to the psychoses, neurasthenia, and hysteria, and also any local or general disease which is latent. The delirium in malaria does not differ in any respect from that evoked by other acute infectious diseases or endogenous or exogenous intoxications. Acute mania may also appear, as well as melancholia.—*Progrès médical*, September 28th, 1901.

ACUTE PULMONARY COMPLICATIONS IN MALARIA.—Crespin and Maillert have investigated some of the lesions present in the lungs in cases of malaria, more especially the broncho-pneumonic (*Arch. Gén. de*

Méd.) They find that acute bronchitis is frequent in many cases of malaria, and that the bronchial lesion is most marked in the bases, especially on the left side. The bronchial complication seems to be in proportion to the amount of lesion present in the spleen and liver. Pulmonary congestion is also common, but varies very much in its intensity in different cases. Pneumonia is also present in several cases, and the prognosis is extremely uncertain, death taking place in a large number. Pneumonia may come on after the malarial attack, and supervening in cases of malaria of long standing is very serious. The temperature in malarial pneumonia is deceptive, and in many cases of post malaria there may not be a very high degree of pyrexia. In fact the writers describe an apyrexial pneumonia of a very serious type. Pneumonia is due to the pneumococcus, not to the hæmatozoa. There is therefore nothing specific in this form of pneumonia. The relation of this latter to the pneumonic process is not quite easy to understand. It is suggested by the writers that its tendency to cause congestion creates a predisposition to a pneumococcal infection. The writers also draw attention to the fact, which has been several times noted, that in malaria there may be an apical consolidation simulating phthisis in many of its characteristics. A curious point about the apical consolidation met with in malarial cases is its tendency to pass to the opposite apex with extreme rapidity. In twenty-four hours the upper portion of one lung may have completely consolidated. The treatment of all these complications is the free exhibition of quinine. This does not, however, mean, according to the authors, that the pulmonary condition is a specific one. The action of quinine is widespread and seems to reduce congestion in the organs generally, and therefore favours the resolution of the pneumonic process.—(*Brit. Med. Jour.*).—*Ind. Med. Record*, Nov. 13th, 1901.

BENEFIT OF INTRASPLENIC INJECTIONS IN CHRONIC MALARIA. By J. L. Castro Gutierrez.—The writer relates several cases of chronic malaria with hypertrophied spleen, absolutely rebellious to quinine or any medicinal treatment. He injected directly into the spleen a Pravaz syringe of a mixture of 20 cg. of malate of iron and six drops Fowler's solution, in water. The evening of the same day an acute malarial attack occurred, with fever at 40 C. Subcutaneous injections of quinine were made repeatedly. The third day the intrasplenic injection was repeated, but was not followed by any reaction, and the patient's further recovery was rapid. The experience was repeated in every case. The malaria parasites had probably ensconced themselves in the spleen, and quinine did not reach them. The intrasplenic injection roused them up and forced them into the general circulation where the quinine administered immediately afterward soon destroyed them. The author asserts, therefore, that the technique should be first the intrasplenic injection, and then large subcutaneous doses of quinine, as soon as the acute attack appears.—[*Chronica Medical (Lima.)*]

SUBCUTANEOUS INJECTIONS OF QUININE IN MALARIAL FEVER.—The great objection formerly alleged against

the hypodermic injection of quinine was the great liability of causing an abscess at the seat of injection; at the present day such an abscess would infallibly be attributed to the use of a dirty needle, and would be looked upon by most physicians as a very unfortunate accident. Dr. Ant. Guiseppe Cipriani, in *La Medicine Scientifique*, however, maintains that such abscesses are beneficial, and he causes them intentionally, it appears, by injecting into the deeper layers of the dermis, instead of into the subcutaneous tissue. His observations, which he says have been verified by others, go to show that by this method the fever is cut short much more quickly than when quinine is given by the mouth or subcutaneously, that the general health is more quickly restored, and that the patients, though living under malarious conditions, keep free from fever a longer time than their neighbours. He has chiefly treated children in this way; the drug he uses is the bichloride of quinine.—*Indian Medical Record*, October 16th, 1901.

A CASE OF MALARIAL NEPHRITIS, WITH MASSING OF PARASITES IN THE KIDNEY.—James Ewing states that microscopical examination of the kidneys of fatal cases of malaria has yielded evidence of three main types of acute renal lesions occurring in this disease: (1) Acute degeneration of toxic origin, often reaching a degree in which exudation of blood serum into the tubules is added. This lesion is responsible for the vast majority of the cases of albuminuria in malaria. (2) An extreme form of acute degeneration, with focal necroses, numerous hæmorrhages, and exudation into the tubules of blood serum and blood pigments. This lesion is seen in cases of hæmoglobinuric malarial fever, and it has not yet been found associated with an excessive number of parasites in the capillary vessels. (3) Massing of parasites in the renal capillaries, with extreme degeneration of parenchyma cells, multiple hæmorrhages, and exudation of blood serum into the tubules. It seems certain that this type of lesion can occur only in severe æstivo-autumnal infections. There is anatomical evidence that in the pernicious æstivo-autumnal cases the three types of lesions may be variously combined, but no good reason for believing that with the benign tertian infection occurring in this latitude any other than the first type can exist.—*American Journal of the Medical Sciences*, October, 1901.

QUININE AND ITS ESTERS.—Dr. M. Overlach says that saloquinine, the ester of quinine, has these advantages over the ordinary preparation of the drug: (1) It is absolutely tasteless; (2) its use is followed by no ringing in the ears or disturbance of hearing, by headache, vertigo, or other symptom on the side of the nervous system; (3) no irritation of the gastrointestinal or genito-urinary tract has been noticed, even after the employment of large doses. It is given in doses of 30 grs., once or oftener daily, to adults. A neutral salt of the ester is equally useful in annulling pain. It is given in increasing doses, from 15 grs. up to 60 grs., omitting the drug for a day when the maximum dose is reached. The author has found it useful in all kinds of rheumatic affections, neuralgia, neuritis, the lancinating pains of tabes, and gonor-

rhoeal rheumatism.—*Centralblatt für innere Medicine*, August 17th, 1901.

EUQUININE IN MALARIA.—The value of the euquinine as a prophylactic in malarial fever, and in guarding against relapse of the fever, is admitted. The Italian observers Celli, Di Mattei, and Mori regard it as of marked prophylactic power. In the intermittent fevers in this country it is of value combined with arsenic and iron.

The following is a practical formula:—

| | | |
|---|--------------------------|---------------------------------------|
| R | Euquinine | grs. ii.—iv. |
| | Acid arseniosi | grs. $\frac{1}{100}$ — $\frac{1}{50}$ |
| | Ferri sulph. exsic. .. . | grs. $\frac{1}{2}$ —i. |

M.—Ft. caps. No. 1. S.—Use thrice daily.

—*Clinical Review*, September, 1901.

TREATMENT OF MOSQUITO BITES.—Dr. A. Manquat states that the most successful treatment consists in the local use of formalin, tincture of iodine and alcohol. He uses the pure formalin or alcohol in one-half strength. The tincture of iodine is objectionable because of the stain it leaves on the skin. As a prophylactic against mosquito bites the following combination is of service:—

| | |
|------------------------|-----------------------|
| Oil of tar | 6 drms. |
| Olive oil | 6 drms. |
| Oil of pennyroyal .. . | $\frac{1}{2}$ oz. |
| Spirit of camphor .. . | 3 drms. |
| Glycerine | 2 $\frac{1}{2}$ drms. |
| Carbolic acid | 1 drms. |

M.—Apply on retiring.

—*Jour. Am. Med. Association*.

PLAGUE.

INOCULATION AT POONA.—From July 1st to December 2nd 719 cases of plague occurred at Poona, of which number 541 died. Fifty-nine inoculated persons had been attacked by plague, of whom only six died. These numbers bring the benefits of inoculation very prominently to the fore.

HAFFKINE'S SERUM.—The statistics of the plague in Bombay seem to have established the fact that Haffkine's plague prophylactic is an effective serum for immunising patients who have been exposed to the disease. Out of 3,814 inoculated persons in the Khoja Mussulman community but three deaths occurred, while among 9,516 uninoculated persons fifty-nine deaths occurred.—*Medical Record*, November 2nd, 1901.

The belief that plague infection in Europe is nearly entirely carried by rats should call for the extermination of these rodents both in ships and at all seaports, if nowhere else. Fighting with rats is not like fighting with anything so minute as the mosquito, and if this latter is not regarded as an impossibility, certainly rats should have but a small chance of existence. There are places where the presence of rats is regarded as a matter of course, though they may not be exactly popular. Now they are shown to be a danger to the community, and their extermination must be regarded as an absolute duty. With their disappearance it may transpire that some other scavengers may have to be employed, for even rats have their use.—*The Hospital*, November 30th, 1901.

THE PLAGUE.—It is officially reported that the plague is at an end in Naples. There were but 15 cases in all, and the time from the discovery of the first case to the discharge of the last from the hospital was but three weeks. In Egypt there were 160 cases during a period of six months, only one of these being in the person of a European. In Rio de Janeiro and Campos, Brazil, the epidemic continues, two or three new cases being reported daily. The representative of the United States Marine Hospital Service at Constantinople has made a report on the plague in that city, which he says has been prevalent since last April. He reports that 20 cases have occurred, although the local authorities, following the example of the Sultan, have shown much willingness in taking necessary preventive steps. The disease has been of a mild type as compared with the epidemic in China and India.—*Medical Record*, November 2nd, 1901.

RAT PLAGUE.—Edington (*Centralb. f. Bakt.*, June 27th, 1901), who has been making investigations at Cape Town, has isolated from a rat, supposed to have died of plague, a bacillus resembling the plague bacillus in cultures and in its effects on guinea-pigs, but differing from it morphologically and in having no effect on rabbits. He concludes as a result of these experiments, that the so-called rat plague is not the same disease as bubonic plague, and suggests that in future in cases where rats die from a disease resembling plague, other animals besides guinea-pigs should be used for diagnostic experiment. The disease produced by the organism isolated by the writer resembles in guinea pigs that produced by the true plague bacillus.

DETECTION OF PLAGUE BACILLUS.—The plague bacillus can be detected during life if an infected gland is pierced with a hypodermic needle, and some of its contents used to make cover-glass preparations in cultures. Novy (*American Journal of the Medical Sciences*, October, 1901) failed in one attempt of this kind because his needle did not actually pierce the small gland in which the plague bacilli were active.

YELLOW FEVER.

A SYSTEMATIC study of yellow fever is about to be undertaken by the Marine Hospital Service, the incentive thereto being the discoveries made by the army medical officers in Cuba, and the demands, based upon these discoveries, made in the interests of commerce for a relaxation of the existing quarantine regulations. For the better carrying out of the desired investigations, an institute will be established for the purpose of collecting all facts concerning yellow fever, to designate the specific lines of enquiries to be made, and to make the same. The work will be divided among four sections, on history and statistics, etiology, transmission, and quarantine and treatment, respectively. The following are the topics to be considered by each of the sections:—

A. History and Statistics: (1) The early history of the disease. (2) Relation to the slave trade. (3) History of recent epidemics (since 1850). (4) Relation to modern sanitation, especially paving, drainage, &c., in cities. (5) Why did not New Orleans have it in early times while Boston did? (6) Mortality statistics.

(7) Maps showing yellow fever zones. (8) Maps showing the infectible territory in the United States.

B. Etiology: (1) The cause of the disease.

C. Transmission: (1) The transmission of the disease by the mosquito. (2) Can any other mosquito than the *stegomyia fasciata* carry the infection? (3) Is the progeny of the mosquito also infected? (4) How many generations? (5) Can the mosquito become infected by any other means than by sucking the blood of a patient sick with the disease? (6) Can the mosquito become infected by contact with the dried-blood discharges or other infected materials upon fomites? (7) Can the disease be transmitted by any other means than through the mosquito? (8) Can the disease be conveyed by fomites, or through the air, soil, or water? (9) The geographical distribution of *stegomyia fasciata* in relation to the disease. (10) Is the immunity enjoyed by certain localities due to the absence of this variety of mosquito? (11) A study of the life and habits of the *stegomyia* and allied species, especially with a view to their extermination.

D. Quarantine and Treatment: (1) Is disinfection of baggage necessary to prevent the spread of the disease? (2) Is any treatment of baggage necessary? (3) Mosquitos in baggage, in merchandise, in cars, in ships. (4) Treatment of the patient. (5) Guards against mosquito bites. (6) Immunity of individuals, of races. (7) Individual prophylaxis. (8) Communal prophylaxis—sanitation.

THE EFFICACY OF QUARANTINE AND FUMIGATION IN THE PREVENTION OF THE SPREAD OF YELLOW FEVER, WITHOUT MOLESTING THE MOSQUITO.—Joseph Waldauer advocates quarantine that will permit commercial intercourse with as little interruption as possible, commensurate with safety. He cites a number of instances, one of which is as follows: In 1897 yellow fever had infected the eastern part of Clinton, Miss. A cordon was placed about the infected centre, and not a single case appeared outside of this boundary. The mosquito was there, but seven-eighths of the population escaped the disease. He reports instances in which he believes fomites are alone responsible for the existence of the disease.—*American Medicine*, October 5th, 1901.

THE PREVENTION OF YELLOW FEVER (by Dr. Walter Reed and Dr. James Carroll).—Referring to the disinfection of cargoes, the authors point out that the only possible excuse for subjecting a cargo to disinfection would be the fear of the presence of infected mosquitoes in the vessel's hold. If the voyage has consumed more than five days, however, all of the mosquitoes contained in the hold will have died. With our present knowledge of the propagation of yellow fever, personal baggage should no longer be subjected to disinfection, and with our increased ability to prevent its spread by measures easy of application, instances should be few and exceptional when a vessel coming from a yellow fever port should be delayed longer than is necessary to remove her non-immune passengers who have not yet completed their period of five days since leaving the port of departure. A most important work will have been performed if we can persuade the sanitary authorities

of Mexico and of the Central and South American States to join us in the adoption of more enlightened methods for the suppression of this widely prevalent epidemic.—*Medical Record*, October 26th, 1901.

ON THE MODE OF TRANSMISSION OF THE INFECTIOUS AGENT IN YELLOW FEVER AND ITS BEARING UPON QUARANTINE REGULATIONS (by Dr. A. H. Doty).—The author does not believe that Dr. Reed's experiments have shown conclusively that there may not be some other means than the mosquito by which yellow fever is transmitted. Considering these experiments, however, in connection with the results of our practical experience, it is clearly evident that this disease is not contracted by personal contagion or through the medium of clothing, bedding, cargoes of vessels, &c. He believes that we are justified in changing our quarantine regulations to conform to these views, and that such a proceeding is safe and reasonable. If the future shows that there are other means of infection, it will be then time enough to add whatever restrictions are necessary for the protection of the public health.—*Med. Record*, October 26th, 1901.

REGARDING YELLOW FEVER ON THE "ETHEL-BRYHTA."—This vessel reached New York on August 21st, one of the crew having died the previous evening. The appearance of the body, the history of the case, and the result of the autopsy justified the Health Officer in officially declaring it to be yellow fever, although the *post-mortem* evidence was not entirely conclusive. Three of the crew, whose temperatures were above normal, were removed to Swinburne Island for observation. These were afterward discharged well, having had no symptoms of the disease. The vessel was disinfected and proceeded to New York, and left there on August 25th, and went to Delaware Breakwater, Norfolk and Jacksonville, arriving at the last place on September 3rd. The vessel remained there (Jacksonville) for about two weeks and then sailed for Santiago, reaching that port on September 19th. On arrival it was found that three of the crew had been sick with fever, and one died; and very soon afterward eight or ten more were affected in the same way. These were at once declared by the authorities to be cases of yellow fever. It was this condition which was reported from Santiago, and which would convey the idea that the disease had been stringing along since the departure of the *Ethelbryhta* from Progreso. Those who have given this matter careful consideration are perfectly satisfied that yellow fever is not transmitted by personal contagion or through the medium of clothing, bedding, &c., and that secondary cases do not occur after five days' removal from the infected area. No one believes that the wife of the captain died of yellow fever, and her death has been satisfactorily accounted for. She was perfectly well when she left Norfolk, according to the statement of the Health Officer of that place. On the following day she was taken suddenly ill and died. The reason of this has been accounted for. Vessels which loaded at the same dock with the *Ethelbryhta* in Jacksonville were found to have a number of cases of fever on board at practically the same time that the report came from Santiago regarding the *Ethelbryhta*. The health officials of Jacksonville have examined these

cases and have declared them to be malarial fever, clear and well defined, and the examination of the blood has shown the plasmodia. Comment is unnecessary. It is difficult for an experienced sanitarian to believe that the cases reported there are those of yellow fever, particularly as the latter disease does not exist in Jacksonville.—*Med. Record*, October 5th, 1901.

PARTIAL FAILURE OF ANTIVENENE.—Dr. Calmette, of the Pasteur Institute at Lille, was recently, as we noted at the time, bitten by a cobra during the course of some experiments he was making. He immediately injected subcutaneously a dose of antivenene which had been prepared by himself, and his life was preserved. He was not saved, however, from all the consequences of the injury, for it was found necessary three weeks later to amputate the bitten finger, on account of gangrene, which set in soon after the accident.

PRIZE FOR FILTER FOR STREET STANDPIPES.—A prize of 3,000 pesetas, or about £85, is offered by the Spanish Government for the best model of a filter adaptable to street standpipes, in order that the public may receive water in the most drinkable condition. The filter must be susceptible of being attached to the standpipes or taken off if required, and must be capable of filtering a quantity of water of not less than 800 litres per hour. A second prize of 1,000 pesetas, or about £28 9s. is offered for the second best model.—*Indian Engineering*, September 21st, 1901.

VACCINE LABORATORY FOR BURMA.—The Superintendent-General of Vaccination, Burma, says that to ensure success in manufacturing lymph, a laboratory must be constructed on sanitary principles on the same lines as a modern hospital. Plans of such a building are being drawn up by the P. W. D. and the necessary plant has been ordered from England. Meiktila is spoken of as a favourable station for the construction of such a Vaccine Laboratory, which promises to be of great use in the province. It is close to the Shan States, where the Chiefs are greatly in favour of vaccination, and has a cooler climate than Rangoon.—*Indian Engineering*, October 19th, 1901.

LIVINGSTONE COLLEGE.—The Annual Public Meeting of Livingstone College, which has previously been held at the end of November, has been deferred until next summer, in order that it may be held in the beautiful grounds of Livingstone College. The Annual Report has, however, been presented to a General Meeting of Members of Livingstone College, incorporated 1900, and will be published shortly, together with other interesting matter, in the Livingstone College Calendar and Year Book. An advance copy of the Report and Financial Statement has been issued to subscribers and other friends of the College, from which the following particulars may be obtained. It will be remembered that the new premises of the College were opened in June last by Dr. Livingstone's elder daughter, Mrs. Bruce, accompanied by her

sister, Mrs. Wilson, and other members of the Livingstone family; work had, however, been carried on at Leyton since the previous October, so that the first complete session in this house has now been completed. The result is considered by those responsible for the administration to be an unqualified success. The premises have proved to be admirably suited for both the accommodation of the students and for the Principal's private residence, and the hopeful forecast which was made a year ago of the advantages which would be gained by the acquisition of Knott's Green House has been fully proved. During the last term 19 residents and 9 non-residents were going through the course of training, a larger number than had ever previously been together at the College at any one time. Unfortunately, this number has not been maintained in the new session, and consequently a special six months' course is being arranged, commencing from January, 1902, which it is hoped may suit some students who wish to avail themselves of the training of elementary Medicine and Surgery which is given at the College.

Turning to finance, the Report states that the General Fund, which includes all the work done in connection with Livingstone College (except those departments which are dealt with under the head of the 'Travellers' Health Bureau), shows a balance of £13 1s. 8d.; but it is pointed out that there still remains a debt of over £3,000 on the property, which will form a serious embarrassment to the work if it is not cleared off. Seeing that this property has been dedicated as a memorial to the late Dr. Livingstone, it is felt that this sum should be fully subscribed, and an appeal is made to all who are interested in the life and work of this great missionary and traveller, as well as those who are concerned with the welfare of Missionaries, to contribute the sum which is still required. With reference to the subject of the Travellers' Health Bureau, it is pointed out that there is a deficiency of £296 18s. 3d. Under this head the Report draws attention to the large amount of work that has been done in connection with the Bureau during the past two years. "Climate," its official organ, has been circulated in all parts of the world, and has made available for ordinary travellers the important results which are being achieved by the schools of tropical medicine, and in other ways has supplied to travellers and missionaries information concerning the various hygienic precautions which should be taken by those travelling or residing in tropical climates. In addition to this, two Exhibitions have been organised by the Bureau under the title of the Livingstone Exhibition, the last, in June, 1901, under the patronage of H.R.H. the Princess Christian, and under the presidency of the Right Hon. Sir George Taubman-Goldie, and having the support of the chief Government offices which are concerned with foreign parts.

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Archives Russes de Pathologie, de Médec. Clinique et de Bacteriologie.

Australasian Medical Gazette.
Boletín de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Brooklyn Medical Journal.
Caducée.
Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Esercito.
Hong Kong Telegraph.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
Journal of the American Medical Association.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Medical Record.
Medical Review.
Merck's Archives.
New York Medical Journal.
New York Post-Graduate.
Pacific Medical Journal.
Polyclinic.
Public Health.
Revista de Medicina Tropical.
Revista Medica de S. Paulo.
Sei-i-Kwai Medical Journal.
The Hospital.
The Northumberland and Durham Medical Journal.
Treatment.

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1.—All communications will be acknowledged in the JOURNAL under the heading "Letters and Communications Received." Contributors who do not see their names in the list should communicate forthwith with the Editors or Secretary.

2.—Manuscripts sent in cannot be returned.

3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.

4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.

5.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.

6.—Correspondents should look for replies under the heading "Answers to Correspondents."

The Journal of Tropical Medicine.

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In order to meet the constant enquiry for addresses of respectable firms catering for the various requirements so difficult to obtain abroad, we give a list of names and addresses which we trust will be found useful to our numerous correspondents and subscribers.

Original Communications.

NOTES ON THE FIRST PLAGUE EPIDEMIC AT CHANGPOO, FOKIEN, SOUTH CHINA.

By J. PRESTON MAXWELL, M.B., B.S., F.R.C.S.

As will be readily seen on reference to the sketch-map provided, Changpoo is the principal city in a large area of country to the south-west of Amoy. Its inhabitants number about 20,000, and the foreign community is entirely missionary. Thus we have here to deal with the spread of disease amongst a pure native community, free from European innovations in the way of steamer or railroad communication.

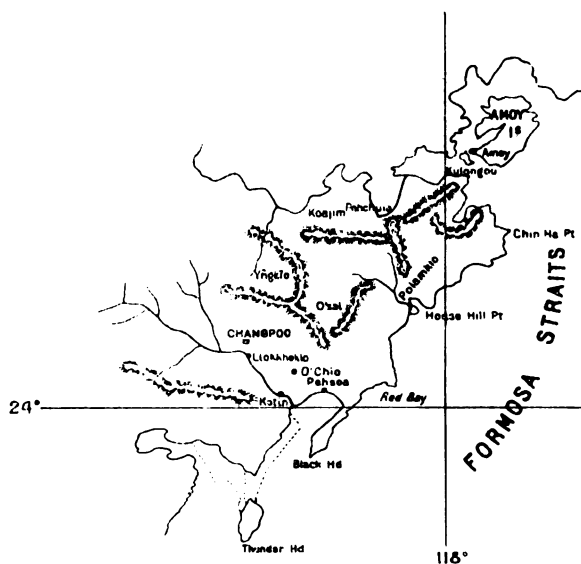
As to the map provided, the coast-line is taken from the Admiralty charts, and the inland places are assigned their position partially by reference to a local missionary map and partly from the knowledge acquired by the writer in travelling over the region.

With these preliminary notes, let us pass to the consideration of the subject in hand. Let me remind my readers of the past history of the disease.

Western China is held to have been an infected place for hundreds of years. But the beginning of the plague of modern times took place in the region of South Kwantung. Thence it was carried to Canton and Hong Kong in the year 1894. Shortly afterwards Amoy became infected.

It is very interesting to trace its subsequent course. Let me direct your attention to the map. There are two main routes whereby Changpoo may be approached. The *first* is *via* Pechuia, Koa-jim, and Têngkio. From Amoy to Pechuia the journey is undertaken in large junks, many of them infested with rats, and of which a large number are engaged in mixed trade. The journey takes but a few hours, and, as might have been expected, shortly after the plague broke out in Amoy, rats and subsequently men succumbed at Pechuia, and plague has been

endemic there since that time. From Pechuia to Koa-jim one travels by small flat-bottomed boats, and the trip takes several hours. The plague appeared in Koa-jim for the first time in 1900, and was preceded by the usual rat mortality. It was not very severe and ceased about the end of July, to reappear in a severer form in the month of May, 1901.



Between Koa-jim and Têngkio, the journey must be performed on foot, or in sedan chair, and several high plateaus have to be crossed. There is a river, but it is exceedingly shallow, and almost unnavigable. From Têngkio, where the plague has not yet appeared, to Changpoo, one meets with the same description of road, but there is a high and difficult mountain pass to be crossed.

The *second* route is by sea from Amoy to Kotin. Large junks, some of them rat infested, go round

laden with kerosine oil, food stuffs, &c. From Kotin they can ascend the river to within four miles of the city, and flat-bottomed boats can go right up to the city gate when the water is high.

There is one other trade route which must be taken into account. In the region around Pehsoa are a scattered set of villages whose inhabitants are fishermen, and who send large junks down to Hong Kong with fish.

As I have before stated, in 1894 plague appeared in Pechuia. This place is almost on the sea-level and to strike further inland a high mountain pass must be crossed. The significance of this will be seen later on.

The second place in which plague appeared, was the set of fishing villages around Pehsoa. In 1896 several men were brought home suffering from plague (from Hong Kong) and died shortly after arrival. Immediately on the back of this, a rat plague commenced, and rats died off in numbers. From this time the plague spread into all the villages in the neighbourhood and also in O-chio, the human mortality being preceded by a severe rat mortality in every village attacked. Then, as the end of June approached, the disease died down. Next year (1897) Kotin became affected. I cannot gather any evidence to determine whether this epidemic was due to a spread from O-chio (about six miles away) or to infection carried by junk from Amoy. In any case it has appeared in both places every year since that time.

In 1897 the disease does not appear to have spread further.

In 1898 O-chio was more severely visited. A Changpoo man, who had been there gambling, was brought home to the city, suffering from plague and died, but the disease did not spread.

In 1899, about the end of May, plague started in a house in the centre of the South Street, Changpoo, and about fifteen persons died. There was also a large rat mortality in this area and the plague was spreading, when there came two or three days of tropical rain. The city streets were flooded several inches deep and the plague ceased to spread. How the disease was introduced on this occasion I have no idea, and can obtain no information on the point.

In 1900 there were no cases of plague in the city as far as I can find out, but towards the close of the year it broke out badly at Liok-khe-kio, a small village on the river about two and a half miles from Changpoo, and there was a considerable mortality.

In 1901 the first real epidemic occurred in Changpoo city.

I have made careful enquiry in all directions, and have carefully sifted all information before accepting it as true.

The plague began in the house of a fishseller, (who later on came under our hands for treatment), near the West Gate, which admits travellers from the Kotin and Liok-khe-kio regions. First, a rat or two died in the house, then the fishseller's wife was seized with bubonic plague (right groin) and died after a few days' illness. This fishseller used to go down twice a week to Kotin, where plague was endemic, and bring in baskets of fish for sale in the city, both salted and fresh; this fish on arrival was sorted and in some

instances washed in the house. The time was the first week in April. Within the course of the same week the rats began to die in houses immediately adjoining, and shortly after there were many people attacked in the immediate neighbourhood. At the same time rats began to die in other houses and shops in the city, and as far as I could learn the rat death preceded the inmates attack about a week; but the time was not absolute.

In all the earlier cases which I attended there was no difficulty in obtaining a history of the previous death of one or more rats in the house. As the epidemic progressed, this was not so easily done, and the people became very reticent on the subject. The plague spread with great rapidity, and it is computed that 1,500 out of the 20,000 died before the 25th of June, and 2,000 or thereabouts fled the city. On the 15th, 16th, and 17th of June rain fell in torrents and almost continuously, and the whole of the city was well washed out, parts of it being flooded. The plague immediately declined and was practically finished in a week.

Consider for a moment or two the method of infection and spread of plague. As to the cause of plague there is no doubt, for the micro-organism can be easily isolated and identified. But how is it introduced into the body and carried from person to person?

Putting aside for the moment the pneumonic cases, which I am inclined to believe form a class by themselves, one is left with the bubonic and septicæmic cases, which form the vast bulk of the attacks. And one is at once struck, and this in company with other observers in different parts of the globe, with the remarkable conjunction of the rat mortality followed by human mortality. So much is this the case that the Chinese term for the plague is "the rat plague," and they know as well as the doctors the connection between the two.

INFECTION BY THE BITE OF A RAT.

But how does the poison get transferred from the rat to man? Sometimes by the direct bite of the infected rat. This is rare, but the following case will serve as an example:—

A man was awakened one night by a rat biting his head. It drew blood. When he had struck a light he found the rat dying close to his pillow. It was at once removed. In twenty-four hours he was seized with plague, and in another twenty-four hours was dead, there being no sign of a bubo before death took place.

RAT-FLEAS.

It has been suggested that the rat-flea acts as a carrier for the micro-organism. Certainly there is every probability of this proving to be true. One afternoon I was called to see a rat which was dying of plague in the court of one of the Chinese houses in which some of our servants lived. When I reached the spot the rat, a fine, medium-sized one, had just died, and the stones all round were dotted with fleas, which were rapidly deserting its body. I did not approach nearer than two and a half feet, and then only to sluice the rat and surrounding region with kerosine, but in that short time I acquired two specimens, which jumped on to the bottom of my white

trousers. Altogether I counted off that one rat *thirty-five* fleas.

Granted that this flea can bite man, a matter which has been denied, one can easily realise how one rat may spread disease, and how eleven members of a family may all die in a single week from the disease; and this theory fits in with and explains cases like the following:—

(1) There are two adjoining villages about two and a half miles from the city. To both villages cases of plague were carried from Changpoo. In the one six cases were thus introduced; all died, yet the plague did not spread. Why? One fact is striking, the rats (of which there were plenty in the village) escaped infection, and there was no rat mortality.

Contrast the fate of the companion village. Here cases were likewise introduced and died but shortly after their introduction. Rats began to die in the neighbourhood of these cases, then scattered throughout village, and cases of the disease quickly occurred.

(2) In another village, about a mile outside the city, rats began to die in the house of a stone-cutter who had not been out of the village for two months. In a week more he was attacked with the disease and died. His house was the nearest in the village to the main highway.

(3) A woman, on rising in the morning, found a rat which had died during the night close to the head of her bed. In a day or two she was taken ill with plague and a cervical bubo developed in the more superficial upper cervical glands and on the side next the place where the dead rat had lain. It was impossible to identify any special insect-bite in this area, but the occurrence is suggestive.

But how does the rat become infected in the first place? My own opinion is, *by means of infected food*. As is well known, rats are voracious and by no means dainty feeders—all is grist to their mill.

Take the case of the fishseller who may have brought home an infected load of fish from Kotin. It is washed and sorted at home, but during the night the rats got at it and start the ball rolling, the disease spreading like wildfire from rat to rat without the necessity for a fresh infection.

FOOD INFECTION.

As to how the food gets infected it is doubtful, but there are many open avenues.

(1) I have seen men, with and without suppurating buboes, handling fish, vegetables and cakes, and even selling the same on the streets.

(2) A pneumonic case is another source of danger. I have seen such a one expectorating all over the room with its earthen-floor and the fowls and dog eating up the sputum.

(3) A third source of infected food is the bodies of those who have died of plague. Many instances of corpse-eating by rats could be put on record, and I know of several cases where plague bodies have been gnawed by them. During the present year (1901) a plague body was buried hastily in a mat in some ground near a missionary institution, which stood some distance away from other Chinese houses. It was found that the grave had been entered by rats, and they were seen leaving its vicinity. In a day or

two rats began to die in the portion of the institution nearest to the grave, and a small outbreak of plague occurred.

DID RATS INFECT CHANGPOO?

But it may reasonably be suggested that the Changpoo epidemic was due to an immigration of infected rats from the outskirts of Liok-khe-kio to the city. There is no doubt that rats do migrate from unhealthy to healthy places. Our own mission houses may serve as proof. Standing in a fine compound, shortly after the plague began, we were troubled with these nightly visitors, who in numbers got into our ceilings and raced around the verandahs at night. On one night there was a large number of these on the verandah who made a deliberate attempt to get in through one of my glass window-doors which was closed. With the cessation of the epidemic these entirely disappeared, presumably having gone back to old haunts.

But the evidence against the theory of the infection of the city is weighty.

Firstly, there is a large suburb outside the West Gate, which should have been first attacked, but was not affected till long after the city.

Secondly, if there had been an invasion of infected rats, one would have expected a much more general outbreak, whereas during the first ten days the disease was localised to a few houses. In some villages, as at O-chio, the disease was certainly carried from village to village by the rats, and it will be noticed by reference to the map that, whatever be the reason, the course of the disease has been on the level, and that where roads had to pass over mountain passes, as to Tâgkio, O'sai, or inland from Pechuia, the disease has not spread in these directions, although there are regular trade routes over these passes.

But passing from the share taken by the rat in spreading the disease, are there not other channels of infection? A second channel of infection is directly by means of infected food. In a village which is as yet uninfected, a woman, whose sole connection with the city is that some of the food she eats was brought from there, develops an attack of plague with a tonsillar gland bubo. There was no doubt about the diagnosis, and although very ill she pulled through. Instructions were obeyed and the village has not had another case of plague. There has been no rat mortality there.

As I have previously stated, the people are extremely careless in this respect, those recovering from plague handling and hawking food: and the flies in Eastern lands are so numerous and persistent in their attentions that they may easily carry infection and so poison the food. As to whether this is a common mode of infection it is difficult to form conclusions. On the whole I think the evidence is against that view.

INFECTION BY AIR-PASSAGES.

A third channel of infection is by means of the respiratory passages. I presume most of the cases of plague pneumonia arise in that way. This would explain the occurrence of more cases than one in the same house with much the same clinical features. The sputum in these cases swarms with the bacilli,

and in some is a pure culture of the same; and it is easy for those who attend on such a patient, who is distressed, and towards the end sometimes fighting for breath, to contract the disease by inhalation as he splutters and spits about the room. Fortunately these cases are not very common.

INFECTION BY THE SKIN.

A fourth method of entry is through a small wound in the skin. I am inclined to think that the existence of this as a separate channel of infection depends on the introduction at the same time of other micro-organisms. If the plague bacillus is introduced alone and in small quantity the skin lesion is probably trifling, but when another poison is superadded you get the following class of cases. A small bulla develops at the site of inoculation. Simultaneously a bubo appears in the glands supplied by this area, or at the least within an hour or two, and the fever also *begins* at the same time. I have seen four cases of this nature. It must be clearly understood, that in these cases the local reaction precedes or is co-existent with the commencement of the fever. In due course the bulla may dry up and form an eschar, or may spread and the skin in this region become gangrenous. These gangrenous patches are distinct from those which come on during the course of the disease and are more of the nature of a pyæmia, as in a case to be narrated subsequently.

But after all is said, one must confess that as yet the exact method of infection in the majority of cases is, to say the least, obscure.

AGE, SEX, AND OCCUPATION.

Age, sex, and occupation, have but little influence on the incidence of the disease. Babies of a month or so old I have known to get it, and old men of 70 to 80 years of age likewise contract it. But the young and strong form the bulk of the cases. A moderate temperature seems to be the most favourable for its development. The Changpoo outbreak began at the commencement of the hot weather, which this year has been unusually moderate, not over 90° in the shade while the epidemic was rife.

THE EFFECT OF FLOODS.

One etiological factor seems to me of distinct importance. It will be noticed that the small outbreak of 1899 and the severe one of 1901 both ceased after the flooding of the city with rain-water. What is the connection? It may be a mere coincidence, but it looks as if the thorough cleansing of the streets, and in many cases the houses also, had contributed to its removal. The underground locations of the rats were probably flooded, many drowned, and the rest driven out. Against this view is the fact that these rats seem to like the rafters and eaves quite as much as their earth or drain homes.

As has been previously noted by other writers, the earlier cases of the epidemic were for the most part most severe, and septicæmic and pneumonic cases were by no means uncommon at the commencement. Death in some cases occurred in from twelve to twenty-four hours of the first symptom.

SIGNS AND SYMPTOMS.

The clinical characters of the disease have already

been fully described by other writers, and I confine myself to a few notes.

As to *premonitory* signs and symptoms, my experience is that, as a rule, they are absent. The disease may, however, be ushered in by malaise of a few days' duration.

In one case there were *distinct* premonitory symptoms. My own horse-boy came to me on a Thursday morning complaining of pain in the glands of both groins. These were palpable and perhaps a little larger than usual, but not enlarged, and there was not a trace of tenderness on pressure. The temperature was normal. On Friday and Saturday he was still complaining, but there was no sign of disease, his temperature was still normal, and he carried 70 lbs. for me fourteen miles on the Saturday. On Sunday, about mid-day, he began to have fever, and on Tuesday buboes developed in both groins. Shortly after nearly all the glands in the body became enlarged, and he died on the eighteenth day of plague, which had become pyæmic.

SKIN RASH.

In three cases I have seen rashes, two of these being purpuric, and one (the case narrated above) pyæmic.

In this case, at the end of the first week papules of varying size developed all over the body, on an erythematous raised base. On the second day they were crowned by a vesicle which grew till the whole papule was transformed into a large bulla. If left to itself this burst, and then an eschar formed on its base, which, separating, left a shallow ulcer. By means of a capillary tube I removed some of the fluid from one of these bullæ and inoculated an agar tube with it, obtaining a pure culture of the plague bacillus.

SCROTAL INFLAMMATION.

In two cases inflammation of the scrotal tissues seemed to take the place of a bubo. In both cases this inflammation appeared on the third day of illness, and was intensely painful. One case died on the fifth day, while the other recovered after a severe illness, followed by a tedious course of suppuration in the scrotum, which involved the testicle and led to the sloughing of a portion of the latter. In both cases the inflammatory process was on the left side.

TEMPERATURE IN PLAGUE.

It is difficult to dogmatise as to the typical temperature of a plague case, but it seems to me to resemble that of a case of smallpox more than that of any other infectious malady. We have the initial high rise, gradually descending on or about the third day as the bubo appears, and rising again for a time as the bubo maturates. It is always a grave sign if the temperature remains steadily high and refuses to fall, and the same may be said of the secondary rise if severe and prolonged. In some cases there is no secondary rise at all, and these are usually mild cases.

CEREBRAL SYMPTOMS.

Stupor or delirium are serious symptoms, especially the former. But a patient who is absolutely unconscious may, under careful treatment, recover. A

delirium which verges on insanity is a sign of almost fatal import. Here are two illustrative cases:—

An old table-boy of mine, aged 23, was seized with an attack of septicæmic plague. When I was called to him about five hours after the commencement, he was in the following condition: His temperature was 105°, and he knew me, and when spoken to firmly, would answer and obey. Otherwise he was sometimes sitting up and sometimes lying down, chattering and laughing to himself. He assured me he was quite well and had no pain, but as soon as his attention was diverted, his face would relapse into its bright smile, and he would act as though he was relating some funny story or listening to the same. In a few hours this passed into stupor, and he was dead within the twenty-four hours.

Another young man, aged 26, was seized with plague. I was called to him at the end of thirty hours. A bubo was then developing in the groin; but he was also wildly maniacal. When I entered he made a dash at me, and was with difficulty restrained by his friends. Then he tore off his upper garment and poured out a string of curses on his friends for meddling with him. After a lot of persuasion I got him to drink his medicine and lie down. But no sooner was my back turned than he was up again and as bad as ever. In a few hours collapse came on, and before the close of forty-eight hours from the commencement he was dead.

CONJUNCTIVAL AND CARDIAC AFFECTIONS.

There are two marked features about an attack of plague which materially aid diagnosis.

One is the suffused injected conjunctiva, sometimes amounting to an actual conjunctivitis. This is more marked than is the case in high fever from other causes, barring severe æstivo-autumnal fever, which can be at once excluded by the microscope; and measles, in which lachrymation and coryza are distinctive features. It is an early and usually well-marked sign. The other is the rapid failure of the heart, as if a direct heart poison were being produced and set in circulation. From the first the pulse is rapid, and in a few hours it may become weak and almost uncountable. Actual attacks of syncope may occur and may be of prolonged duration, but as a rule an attack of syncope is only the prelude to the end.

Here is an exception: I am informed that a man in the North Street, Changpoo, who was suffering from plague, rapidly became worse, finally collapsed, and was supposed to be dead. His relatives procured a coffin, put him in it, and went out to hire bearers. It was a wet night and they could procure no one. Fortunately they did not cover the coffin. At midnight, or some six hours after his supposed death, the man sat up and asked for tea. He ultimately recovered.

One or two more cases of a *subpneumonic* type have been seen. They had fever, blood-tinged sputum, small in amount, but little increase of frequency of respiration, and died after five to seven days' illness. Clinical examination revealed a little broncho-pneumonia, but no striking physical signs.

CASES OF ADYNAMIC TYPE.

Also three or four cases of an *adynamic* type have presented themselves for treatment.

In these cases the temperature keeps low, say, 99.5° to 100°, the patient is collapsed and cold, and the bubo develops slowly. These are mostly patients over middle age, and the mortality is high. One case is interesting as showing the result of playing with the disease.

A man, aged 45, an opium smoker, had been under treatment for eight days and was getting better; temperature, pulse, and appearances improving. He got restive and got up to go and have a smoke at his favourite drug. He had it, came home and lay down on his bed and was dead in half an hour.

TREATMENT.

As to treatment. I have not had any opportunity up to the present of seeing the results of the use of serum as a prophylactic or curative agent. During this epidemic there was no chance of securing a supply, but it should certainly be used whenever possible. Foremost among the remedial agents I should place good nursing. None of the Changpoo cases had any nursing beyond that afforded them by their own friends. But in my own cases even the small amount they secured made a great difference to the chances of the case, and this was specially marked among those who were willing strictly to obey orders.

The mortality rate was about 65 per cent. amongst our cases and was considerably lower than amongst the untreated, of whom, as far as I can judge, about 90 per cent. died. It may be said that the epidemic was a mild one. If the proportion of pneumonic and septicæmic cases be taken as a criterion I think this was not the case.

Pneumonic plague is the most fatal of all the forms and I have never yet seen one recover. Septicæmic plague is the next in point of danger, but a number undoubtedly pull through. Bubonic plague is the least fatal of the three forms.

CARBOLIC ACID.

As far as my observation went, *carbolic acid* in large doses appeared to give the patient the best chance. It brought on profuse sweating and seemed in some way to neutralise the toxin manufactured by the plague bacilli.

As an illustrative case let me quote the following: A young man, aged 26, who had been engaged in burying plague corpses, was attacked with high fever, pain in the right groin, and rapidly became unconscious. I injected 6 grs. of pure carbolic acid dissolved in water hypodermically. Within two hours he had become sufficiently conscious to take another 10 grs. by the mouth. This brought on profuse sweating. In the evening (eight hours after the first injection) he had another 10 grs. of the pure acid. He rapidly improved and his groin bubo, which was enlarging, began to subside. Then he became reckless, got up and had a relapse, which yielded to a repetition of the same treatment, minus the hypodermic injection. It was hoped at one time that carbolic acid would prove to be a specific remedy, but such is manifestly not the case and there are many points about its action which are as yet obscure. Why should it in one case of plague act like a charm and in another apparently similar case have no effect whatever?

Why do these patients not get carboluria? One of my patients *may* have had carbolic acid delirium, but I was by no means certain of the diagnosis, and with the exception of this case I have not seen it.

The best way to administer the drug is mixed with plenty of rice water, which being mucilaginous prevents the irritative action of the carbolic acid on the stomach, and as this can be obtained in any Chinese home it is peculiarly convenient for those who work among this people. If possible, 30 to 60 grs. should be administered in the first twenty-four hours.

GENERAL TREATMENT.

Combined with this treatment, the appropriate general treatment of each case should be carried out. The bowels *must* be kept freely open, and diarrhoea, unless excessive, need not be checked. In the early stages a calomel and jalap purge is very useful.

Digitalis is very useful in sustaining the heart's action. It cannot be spoken of as a specific, but in some cases acts with great success. The same thing may be said of *strychnine*, which in hypodermic injection is often invaluable. But when all is said, one must confess that plague, rather than the physician holds the field. Trustworthy men have, from time to time believed that a specific remedy had been found. At one time it was Yersin's serum, at another calomel, then *digitalis*, and finally carbolic acid. Time alone will show what is the true worth of the pretensions of the latter drug. The best treatment for plague is *prevention*. I am not willing to acknowledge that sanitation and like measures count for little.

PREVENTION.

In Changoo the hospital is touched by Chinese houses on three sides. In each of these houses there was plague, and in one a most virulent outbreak. But the hospital, which was kept rigidly clean, remained quite free, although, in spite of the panic, there were thirty to thirty-five in-patients who were not confined to the hospital. Several cases of plague were brought in, but sent home at once. The Chinese students, eight in number, went regularly with us to the homes of the patients. They took the same precautions as ourselves with regard to careful washing and the use of flea powder, and none of them took the disease. Rats were looked for and hunted down, the dead ones being burnt at once, without handling, by drenching with kerosine and applying a light. There is little doubt that the most important means of prophylaxis is the stamping out of the rat. In some places a severe attack of plague has been noted, once in two years; and this has been attributed to the rats having had insufficient time to make up for the numbers that had perished during the first year.

In some places it might be possible to reproduce artificially the condition which appears to have arrested the plague at Changoo, viz., the flooding of a plague area for at least twenty-four hours with fresh or salt water. In some places this would not be difficult to accomplish.

DENTAL CARIES.¹

By KENNETH W. GOADBY, L.D.S.Eng., D.P.H.Camb.
Dental Surgeon to the Seamen's Hospitals.

To attempt the discussion of all the various theories that have been from time to time advanced to explain the phenomena of dental caries and to point out all the fallacies therewith connected is outside the limits of the present lecture, and I propose to only deal with what seems, in the present state of our knowledge of bacteria, to be the most rational and acceptable explanation.

In the first place the tooth as a tissue is unique, it contains the least amount of organic tissue of any other part of the body, while it is covered with a structure, the enamel, almost devoid of any organic matter at all, the carbonates of calcium sodium and phosphorus having replaced the organic matrix in a manner which may be compared to the formation of fossil wood.

Enamel, therefore, is devoid of physiological reaction to pathological or other stimuli, and we are unable to apply to it the ordinary picture of inflammation as seen in other tissues, including compact bone; it follows that the only explanation that we can properly apply to dental caries is an essentially mechanical one.

THE FIRST POINT OF ATTACK.

In the great majority of cases the first point of attack in dental caries is the interstitial surface of the tooth, the next the crown fissures, and is due, as we shall see later, to the action of acids, produced by bacteria from the food, upon the lime salts of the enamel covering. The enamel at the seat of attack is seen microscopically to have lost some of the cement-substance which intervenes between the contiguous enamel prisms or irregular rods of which the enamel layer is composed. This cement-substance disappears first when a thin slice of enamel is decalcified under the microscope, and at the same time it will be observed that the interstitial substance passes into the enamel rods, producing a structure that may be compared to a series of pill-boxes arranged end to end with a disc of paper between each. These offsets also dissolve away in the acid, and produce, in caries, a great friability of the enamel at the affected spot.

As the process of destruction progresses, the bacteria file into the capillary channels and gradually approach the dentine as their acid fermentation softens and dissolves the enamel rods.

Dentine or ivory, which makes up the large bulk of the tooth, is nearly related to bone; it is permeated with fine canals called dentinal tubules, homologous with the canaliculi of bone, communicating at the enamel surface with the interglobular spaces or lacunae, and at the centre of the tooth with the pulp chamber. Once the dentine is reached a change takes place in the process dependent on the structure with which we are dealing. The dentine matrix is similar to that of bone, and when softened by removal of the lime salt is capable of digestion by the proteolytic

¹ A lecture delivered to the students at the School of Tropical Medicine, London.

enzymes formed by many bacteria. Coincident therefore with the decalcification of the dentine, digestion takes place by the enzymes of certain bacteria, most of which are facultative anaërobic. The combined result is a gradual hollowing out of the interior of the tooth until the edges of the cavity break away, revealing the underlying decay.

The bacteria concerned gradually pass along the dentinal tubules and reach the pulp, which already lowered in its resistance by the toxins that have filtered along the canals, succumbs generally with the well-known symptoms of acute toothache.

Further, the blood-vessels which supply the pulp are derived from those of the dento-alveolar periosteum, and infection is conveyed along them to the bone and soft tissues. In this way a large number of the tubercular glands in the necks of children are brought about, the tubercle bacillus either passing in through the route indicated, or else circulating in the blood is brought to some gland already in a state of chronic inflammation as the result of neglected carious teeth, often of the first dentition. The weakened gland is quickly infected.

Such then, is a brief outline of the phenomena of dental caries.

FOOD STUFFS.

We will now consider the process a little more in detail in its earlier stages.

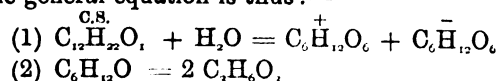
Food stuffs, as you are aware, consist of three main classes. Of these proteids and fats are of little importance in the present connection. Proteids are fermentable by bacteria in the changes of putrefaction with the formation of some of the organic acids such as *oxyphenyl-propionic* acid and *oxyphenyl-caproic* acid. Proteids, however, are only acted upon when all the carbohydrate present has been changed, so that in a diet consisting of a large proportion of carbohydrate such organic acids are present in only the most minute quantities. Very little is known about the fermentation of fat, but probably fatty acids and glycerine are formed to some small extent.

With carbohydrates the case is different, many bacteria attack them and of these bacteria many representatives occur from time to time in the mouth.

Of the three classes of carbohydrates the monosaccharides are the most easily acted upon, whereas the di- and poly-saccharides are not so easily fermentable, but require preliminary inversion either by a ferment produced by the one organism or by another organism working in symbiotic activity with the first.

The change that is effected is the addition of a molecule of water changing the di- to the monosaccharide. The end product is generally lactic acid, although other acids such as acetic or butyric may be formed in small quantities.

The general equation is thus:—



Some of the mouth bacteria seem capable of directly fermenting the di-saccharides, particularly maltose and iso-maltose, an interesting fact when we consider that the chief end-product of ptyalin on starch is maltose; and we must bear in mind the significant facts of the large place that carbohydrates

have in the dietry of those convalescent from various acute diseases, and the frequency with which caries dates from such an illness.

MOUTH FERMENTATIONS.

Turning now to the point of view of the fermentation physiologist we are reminded of the fact that in all natural fermentations, that is those occurring spontaneously in nature and not by art, the class of food stuff in a given solution largely determines the class of organisms present; thus, to take a general illustration, crude wine must, when it comes from the press, contain a vast number of species of bacteria derived from the skins of the grapes, from the workers' hands, the air, and other sources. In the usual course of events, however, alcoholic fermentation takes place. The yeasts present in the medium finding the conditions fitted to their development increase in great quantities, with the result that an alcohol is formed. When the alcohol has become of a certain percentage the action ceases and the advent of another or acid fermentation is ushered in. The bacteria producing this were present in the original, but owing to the alcohol on which they thrive being unformed were unable to develop. This acid fermentation only ceases when all the alcohol present has been changed to acetic acid. A third series of bacteria now come into operation, contained like the previous two series in the original must; these bacteria ferment the acetic acid to CO₂ and water, changing the reaction to neutral or faintly alkaline. The putrefactive bacteria now come to the front and from the remaining proteid present produce the various evil-smelling products which are associated with putrefaction.

Such a sequence of events is termed a "metabiotic cycle," one class of bacteria preparing the way for the operations of another; while when two or more bacterial species work together the process is termed "symbiotic." It is most probable that dental caries is produced in this manner.

So far then, it is evident that carbohydrate food and fermentative bacteria have an important bearing upon the question of caries, and that proteid, that is, meat diet, has little relation to tooth destruction.

CARBOHYDRATES AS A PREDISPOSING CAUSE OF CARIES.

Let us now examine the evidence which points to the special effect of carbohydrates as a predisposing cause of caries.

If the problem were only one of the presence of carbohydrate food it seems strange that any teeth should escape, and with such a proper critical point of view we will proceed in our enquiry.

In the first place, if we examine a section of tooth enamel showing one of the interstitial or apposing surfaces of two contiguous teeth from a mouth in which caries is well marked, we find curious felt-like masses of bacteria attached to the enamel. Under this bacterial sheet the enamel is seen to be in an incipient stage of caries with the destruction of the intercolumnar substance. If now we make a culture from a part of such a tooth we find that there are frequently present organisms which produce a curious thick, gelatinous growth, which is most diffi-

cult to remove from the culture tube. If now we suspend a tooth in a fluid medium containing such a culture we can reproduce the same layer upon the tooth surface.

If a culture tube containing some carbohydrate is now substituted for the previous experiment and inoculated with an organism capable of producing acid fermentation, it is found that the same changes in the enamel are produced that we have seen are found in the tooth in the mouth. Moreover, even if the organism producing the stringy growth is not of itself an acid producer, many other bacteria will grow in symbiotic relation with it and produce the foregoing changes. In media containing no carbohydrate, that is for instance in inosite free broth, no such change occurs. Sometimes the plaque-forming bacteria will form curious dotted colonies upon the surface of the tube, especially when potassium nitrite is added to media. These plaque-forming bacteria are common in carious mouths. In normal healthy mouths entirely free from caries almost the only species present is the *Streptococcus brevis* of van Lingelsheim, whilst when caries is present acid-forming bacteria are in considerable numbers. Our initial hypothesis then shows considerable likelihood of proof, and we may, I think, conclude that:—

CONCLUSIONS.

- (1) Carbohydrate food tends to the furtherance of dental caries.
- (2) Acid-forming bacteria are the ones largely responsible for the process.
- (3) Protein will tend to the reduction of caries.
- (4) Fermentative bacteria of the putrefactive class, in that they tend to produce an alkaline reaction, will also prevent the development of rapid caries.

TEETH OF NATIVES OF THE TROPICS AND EUROPEANS COMPARED.

You are all aware of the remarkable difference that exists between the teeth of the average native and that of the average European. Have we therefore any data in what we have seen to be a logical explanation of dental caries to account for this extreme difference, so great in fact that in Europeans the percentage with carious teeth is 87 to 90 per cent., while in the whole of the various native races of which I have statistics it does not exceed 30 per cent.

Firstly, so far as my somewhat limited experience goes, I find that the enamel of native races, that is, Negro and Negretto, as apposed to Xanthrochroi and Melanochroi, rarely shows microscopical defects and irregularities while in Europeans it is seldom possible to find all the teeth free from even macroscopic irregularities.

Secondly, owing to the effects of sexual selection on the type of beauty and hence the gradual establishment of the most admired type, as well as the larger development of the frontal lobes of the cerebrum, the facial angle has considerably altered and with it the superior maxilla has become contracted with concomitant crowding of the dental arch. The teeth have remained for the most part the same size, with the result that irregularities and crowding are common and a very fruitful cause of caries. It is in this species of mouth that caries is so common.

Thirdly, the general physical condition of food stuffs differs; the coarse, hard and fibrous food is no longer used, and its cleansing effect on the tooth-surface is replaced by the soft, smooth variety of food so commonly consumed.

The following table gives the percentage of caries in certain native races; it is by no means complete, and I shall be much obliged for any information that you can give me, derived from personal observations concerning: (1) Caries (a) *temporary dentition*; (b) *permanent dentition*. (2) Food. (3) Habits of mouth cleansing.

The few direct experiments I have been able to make concerning the bacteria present in the mouths of natives tend to show that the flora is nearly related to that of the monkeys from which I have been able to obtain cultures. In all cases examined putrefactive bacteria were present in large numbers, the number of acid formers being small.

TO PREVENT CARIES.

The practical question that arises at the end of our enquiry is, what steps can be taken to arrest caries or to prevent its appearance? Much may be done by proper attention, and such attention should date from the appearance of the first temporary tooth, particularly in hand-fed children. The use of teat comforters should be absolutely forbidden. The mother, as a rule, takes no notice of the teeth that are carious till often all the temporary teeth as well as the first molars have been attacked, and then even, do what we may, the mouth teems with the organisms specially related to caries. The use of the tooth-brush should be taught early, as well as the more efficient "dragon cane," or its equivalent. The following formulæ may be suggested as of general use:—

| | | | |
|--|----|----|---------|
| Prophylactic. | | | |
| Magnesia Carbonis | .. | .. | 3ss. |
| Lysol | .. | .. | ℥ xii. |
| Aq. Rosæ | .. | .. | ℥ iij. |
| 5i. to be used night and morning, and sluced well round mouth. | | | |
| Dentifrice. | | | |
| Creta Prep. | .. | .. | 5i. |
| Pulv. Saponis dent. | .. | .. | ℥ ij. |
| Pulv. Iridis. | .. | .. | ℥ ij. |
| Pulv. os. Sepia | .. | .. | ℥ i. |
| Sodæ Bicarb. | .. | .. | 3ss. |
| Lysol | .. | .. | ℥ x. |
| Saccharine | .. | .. | gr. i. |
| Otto Rosæ | .. | .. | ℥ viij. |
| Mouth wash. | | | |
| Formalin (40 %) | .. | .. | ℥ ij. |
| Aq. Rosæ | .. | .. | 5i. |

TABLE SHOWING APPROXIMATE DISTRIBUTION OF CARIES.

| | PER CENT. |
|---|-----------|
| Esquimaux (diet: meat only) | 1.4 |
| New Zealanders | 3.02 |
| N.W. Coast America (diet: dried fish) | 3.9 |
| North-American Indians (diet: meat and roots) | 4.5 |
| Fiji Islanders | 5.2 |
| Northern India (diet: vegetables and wheat) | 5.9 |
| Eastern Polynesia (diet: vegetables and fish) | 11.4 |
| Southern India (diet: rice, many sweetmeats) | 14.08 |
| Zulu Kaffirs (diet: milk and vegetables, some meat) | 14.2 |
| Sandwich Islanders (diet: vegetables and little meat) | 19.04 |
| Australian (diet: meat and roots) | 20.4 |
| Bushmen (diet: meat, roots, locusts, &c.) | 24.6 |
| African Slaves (diet: mostly vegetable) | 24.6 |
| Tasmanian | 27.2 |
| European | 87 to 90 |



**PHOTOGRAPH SHOWING RESULTS OF "AMPUTATION FOR PERFORATING ULCER
OF THE FOOT IN LEPERS."**

See an article on this subject by Edward Horder, F.R.C.S.Edin., in the JOURNAL OF TROPICAL MEDICINE, Jan. 1st, 1902.

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THE

Journal of Tropical Medicine

JANUARY 15, 1902.

MR. JONATHAN HUTCHINSON'S VISIT TO THE LEPER SETTLEMENT ON ROBBER ISLAND, CAPE OF GOOD HOPE.

MR. HUTCHINSON has gone to South Africa on a self-imposed mission and with a philanthropy of purpose alike admirable and creditable. We may safely say every one is acquainted with the attention Mr. Hutchinson has paid to the subject of leprosy, and most are aware of the beliefs which underlie his opinion as to the cause of the disease. Irrespective of any question of the elucidation of the cause, Mr. Hutchinson's visit is sure to prove of great value to the study of leprosy in general. His name has been associated for so many years with all that is best in clinical and scientific investigation and teaching, that medical literature is sure to be enriched by his visit. We do not think, perhaps we do not hope, that the visit to

Robben Island will alter Mr. Hutchinson's views, either as regards the question of the segregation of lepers, or the part played by eating uncooked fish in the etiology of leprosy. His beliefs are not founded on any ephemeral basis, they are the result of serious thought and close observation, and we cannot conceive that the experiences gained in a leper settlement are likely to upset his conclusions. It must, however, be said that neither of Mr. Hutchinson's contentions have gained many followers. Few, very few, are willing to allow lepers to wander freely amongst a healthy community, and still fewer accept his theory of uncooked fish as a cause. That does not, however, prove that his arguments are wrong. It may be that all Mr. Hutchinson states and believes in regard to the matter is correct, but most regard his contentions as "not proven."

At the present day scientific thought demands more exact information than mere speculative opinion, however well-fitting that opinion may appear to be. Mr. Hutchinson, however, bases, or holds that he bases, his opinion on the surest of all foundations, namely, clinical observation, and clinical observation has been, is, and ever shall remain, the ultimate appeal.

Leprosy being a proved bacillary disease, should fall into line with other similarly originated diseases, and should afford all the evidence which it is necessary to furnish according to the maxims of the bacteriologist. But even should it do so, are we to accept the bacteriologist's word as final? We regret that it should be so, but bacteriology is not yet an exact science, it is only on its trial, and while willing to attribute to bacteriology a prominent place in our means of research, we cannot place it, as yet at all events, higher. Observation of the behaviour of disease, although confessedly slow in attaining to any real exactitude, yet remains our surest and our safest guide to scientific precision, and all our "ologies" must yield to the truths to be gathered from experience and observation.

The whole question, then, of Mr. Hutchinson's dogma turns on the truthfulness of his observations. That they are made with care and open-mindedness, all who know Mr. Hutchinson are

well aware; but even the wisest and most careful of men may fail to interpret their observations correctly. That Mr. Hutchinson has done so is an open question, and one cannot set aside the opinion of this, our greatest clinical observer, by mere empiric statements.

By his visit to Robben Island, Mr. Hutchinson has set an example to the younger and to the older members of our profession; he goes to see for himself, to gather facts on the spot; and whatever his conclusions may be he will be listened to with respect, if not with conviction, and his observations are sure to furnish us with a fresh and reliable source of valuable clinical material.

THE BEST METHOD OF ADMINISTERING QUININE AS A PREVENTIVE OF MALARIAL FEVER.

By EDWARD HENRY READ, M.R.C.S., L.R.C.P., L.S.A.
Lagos, West Africa.

THE best method of administering quinine as a prophylactic depends entirely on the individual, and his or her state of health.

For ordinary purposes we may divide individuals into three classes: (1) Adult male; (2) adult female; (3) children of either sex.

CLASS 1.—If the person is in perfect health there is no simpler or better way than using the tabloid of a soluble salt, preferably the bihydrochlorate. If the digestion is good and the bowels are regular it never fails to be absorbed. If the person is suffering from constipation or dyspepsia the tabloid can never be depended upon and should not be given. In such case the solution is no doubt the best, but as there are very few who do not object to the taste of quinine—which in many people produces actual nausea—it is better to find some other method of giving the drug. As it is only for prophylaxis and not cure, hypodermic and rectal injections are out of the question. If the person is in the habit of taking porridge for breakfast, a very simple way is to mix the powder with the first spoonful of porridge and then swallow. Given in this way the bitter taste is hardly perceptible, and passes away with the next spoonful of porridge. I have persuaded many people to take it in this way when I could not get them to take it in any other. Another good way of taking quinine is in milk. Mix the powder with a tablespoonful of milk and swallow, after having first lubricated the mouth with a little bread and butter; given in this way the bitter taste of the drug is not perceived (*vide* "Tropical Diseases," Manson, 1900 edition, p. 134).

I think that there are very few people who cannot take quinine in one of the above ways.

Dose as a prophylactic.—15 grs. every fifth day; 15 grs. once a week is not quite reliable, and 5

grs. every day I think is too much. For the first three years of my residence on the west coast of Africa I took 5 grs. of quinine every day, and although I practically had no fever, there is no doubt that the daily dose of quinine has had a deleterious effect on my digestion. If the stomach is given four days' rest between taking the larger doses I do not think the stomach will suffer much even if the drug is continued for many years. For the last nine months I have been taking the larger dose, and I have not had the slightest touch of malarial fever, and I have found no parasites in my blood during this period.

CLASS 2.—Adult female. In ordinary circumstances I think that they should proceed in the same course as class 1, except the dose should be smaller—8 grs. to be taken every fifth day.

Women, as a rule, cannot stand as large a dose as men, and suffer much more from the effects of the drug. Again, they do not require it for the following reasons—they are not so robust, have not the same quantity of blood to be saturated with the drug, and are not so exposed to the vicissitudes of the weather.

Pregnant Women.—There is no harm in pregnant women taking quinine as a prophylactic if the dose is given regularly from the commencement of pregnancy. Although quinine does produce abortion in rare cases, I think it is only in women who are prone to abort. In these cases I think that one fever fit is much more likely to produce abortion than a regular course of quinine. I have given quinine to pregnant women in fairly large doses without any untoward result. I think that 8 grs. is rather too large a dose to be taken by pregnant women, and I should recommend a daily dose of 2½ grs. during the whole period of pregnancy.

CLASS 3.—Children of either sex. There is great difficulty in administering quinine to children on account of their repugnance to the bitter taste; also their various ages have to be dealt with, and the dose as a prophylactic worked out in each case. Quinine can be administered by sugar-coated tabloids or by chocolates containing the drug, but I have had no experience with either and cannot say whether the drug is absorbed when given in this way. A fairly good way of getting children to take it is to put the powder into a spoonful of milk, followed by a spoonful of jam or honey. In this way one can often get them to take it and the quinine is certain to be absorbed.

Doses for children as a prophylactic: Under 1 year, ½ gr. every third day. One to 5 years, 1 gr. every third day. Five to 10 years, 2 grs. every third day. Ten to 15 years, 3 grs. every third day. Above 15, the ordinary adult dose.

In summing up the foregoing, I come to the conclusion that the best way of giving quinine as a prophylactic is the following:—

Class 1: For adults in good health, tabloids in 15 gr. doses every fifth day. For adults in indifferent health, the solution, if possible 15 grs. of the salt every fifth day. If solution objected to, 15 grs. of the powder in a tablespoonful of porridge, or 15 grs. of the powder in a tablespoonful of milk, to be taken every fifth day.

Class 2: For adults in good health, tabloids in 8 gr.

doses every fifth day. For adults in indifferent health, the solution, if possible 8 grs. of the salt every fifth day; if solution objected to, 8 grs. of the powder in a tablespoonful of porridge, or 8 grs. of the powder in a tablespoonful of milk to be taken every fifth day.

For pregnant women, 2½ grs. to be taken daily in either the above ways from the commencement of pregnancy.

Class 3: Children of either sex, age under 1 year, ½ gr. of the powder to be taken every third day in a spoonful of milk, followed by a spoonful of jam or honey. One to 5 years, 1 gr. every third day in a spoonful of milk, followed by jam or honey. Five to 10 years, 2 grs. every third day in a spoonful of milk, followed by jam or honey. Ten to 15 years, 3 grs. in milk every third day. Above 15 years the ordinary adult dose taken in the same way.

ON THE BEST METHOD OF ADMINISTERING QUININE AS A PROPHYLACTIC AGAINST MALARIA.

By TERTIUS.

I ASSUME that it may be taken for granted that quinine is a prophylactic. There is much evidence for this, and the discussion on the subject at the British Medical Association meeting in 1900 may be mentioned, the general opinion being that it was useful.

I now, therefore, discuss only the way in which it acts as a prophylactic and the best means of employing it. In the first place I think that there is no doubt that quinine in any dose will not prevent a mosquito from biting the taker of the drug, nor, if infected, from transmitting the organism; so that if, as assumed, it acts at all, it must be by destroying the plasmodium after it has entered the blood.

Now the sporozoites take a certain time to develop into the amœboid form, or to have resulted in sufficient number of the amœbæ to cause an attack of fever, and the organism is probably best attacked by quinine when it is in the amœboid state in the corpuscle; but seeing that the individual, not having had fever, does not know when this occurs, the best method of giving the quinine will be that one by which this stage in the life of the organism will most probably be affected by it.

By giving a small dose every day it is not at all certain that this time will be the correct one, and if this dose were given at intervals during the day it is probable that only one part of it would be of any use; the remainder would be wasted, and the comparatively small dose which happened to have been given at the right time would, as only small doses could be given for this purpose, be most likely insufficient. The difficulty, however, of getting men to subject themselves to a perpetual multiple daily dosage would render it practically impossible.

I imagine that very few would try to kill the organism, when they knew that a man had it in his blood, by daily doses of 5 grs. of quinine; and I cannot see why they should hope to do so—though it may be possible—when they do not know that it is in

the blood. In fact, small daily doses, though they may be useful, seem to me wrong in principle.

If, however, a larger dose could be given so as to be absorbed at the time during which the organism is maturing—a period of some hours' duration—the end would be achieved.

There are three periods of maturation—twenty-four, forty-eight, and seventy-two hours, according to the organism; and if a dose is taken daily it may obviously hit off any of the organisms; but it may be too late or too early, may be eliminated and wasted, while in the case of the seventy-two hour organism two out of every three doses must be wasted; so the best method is reduced to giving it in such a way that, with the greatest probability and with the greatest efficiency the work must be done, on any one of the different forms of organism.

I believe that this result will be best gained by giving fairly large doses on two consecutive days during the week, and that then, if one dose happens to be useless, the other will probably act on both the quotidian and tertian forms, while if either or both of these doses are too late or too early for the quartan, the following week, if corresponding days are observed, will bring the dose right for it also. In fact, I believe that two doses of 15 grs., given on two consecutive days once a week, will produce better results than 35 grs. taken during the week in 5 grs. daily.

The time for giving it is preferably a short time before bedtime, and then any headache or cinchonism will have disappeared during the sleeping hours. Owing to the European habit of observing the Sabbath, both for the white man and his employes, Saturday and Sunday evenings are convenient. Within the last year I have had the opportunity of seeing the experiment of giving the quinine in the above manner tried on a fairly large scale.

Near to where I am stationed there is being opened a large sugar estate, which at first, and for nearly a year, was singularly healthy; but then as the number of coolies, and probably of infected coolies, and their children, became larger, fever commenced and rapidly increased, until at last there was a very large epidemic. The type was not a severe one, crescents were never found, and not a large number died, but the sickness rate was very high. There were about 1,200 coolies, and for some time the admissions into hospital were forty per week—on one occasion there were forty in a day, and thirty cases a day was not an uncommon number to be treated as out-patients for the same disease; the loss of labour was therefore very great.

Dr. Paul, who was then medical officer to the estate, decided to try the plan, and, so far as I know, originated it. The coolies were mustered every Saturday and Sunday afternoon, and each was given 15 grs. of quinine sulphate in solution, though capsule or cachet might be just as good.

The result exceeded his or my expectations; the weekly admission rate stopped almost at once to about twelve, and many of these twelve Dr. Paul was able to show had escaped their quinine ration. There has never been much fever since, occasionally only two or three fever cases in the hospital; and the loss of labour, which at one time was very serious, is now trifling. On one occasion, while I was in charge, I

tried 10 grs. instead of 15, but there were signs of an increase which made the manager anxious to get back to the larger dose.

There has been one slight increase, but it was nothing to speak of, the cause I do not know, and it only lasted a short time.

I would mention that no other preventive measure was adopted.

I think that this experiment may be considered to have been a success, and to have been on a large scale; the manager would not willingly give up the quinine ration, though the cost is large owing to the number of coolies, but he considers the cost is recouped by the extra amount and efficiency of the labour.

The Europeans on the estate have not taken quinine, but have been fairly free from fever, due, I believe, to the difficulty of anopheles in getting infected, while, if they are infected, they will most probably, from the numbers, rather feed on the coolie who will get his two doses of quinine, and be unharmed.

I believe that for Europeans this method would also be the best, especially for those in bad malarial places, those on expeditions, and those whose occupation renders them liable to infection. For large numbers, such as regiments, jail populations, estate or railway construction coolies, I think it would be found to be invaluable.

THE DURATION OF THE LATENCY OF MALARIA.

By A. SIMS, M.D., C.M.(Aberd.), D.P.H.(Birm.); Certified London Tropical School of Medicine.

Late Medical Officer Congo Free State, and Civil Surgeon of French Government (Brazzaville).

THE duration of initial latency after primary malarial infection is easier to account for than the latency observed after a first attack of fever; the former depends upon the number of valid sporozoites trans-stellated by the mosquito, the latter upon degree of acquired immunity. The calculation which follows, to explain the former, presupposes that all the sporocytes, resulting from the injected sporozoites, lived. In most cases all of the spores survive at this initial period. After a first attack of fever it has been shown that this is no longer the case: "the parasites in the first observed cycle were 500, in the second they should have been at least 3,000, in the third 18,000, and at the commencement of the fourth cycle 108,000, it is certain that no such multiplication occurred" (JOURNAL OF TROPICAL MEDICINE, p. 110, April 1st, 1901). One would naturally expect that the spore slaughter would take place now; but it is not so; otherwise the usual incubation period would be longer than five to fifteen days. The mechanism, so to speak, of their destruction is not yet set up; after a first fever it is in action.

From observations and counts done by Dr. Gray and myself (JOURNAL OF TROPICAL MEDICINE, April 1, 1901), at the Seamen's Hospital, I am led to believe that about 400 fully-grown parasites to the c.mm., or the presence of about two billion adult parasites in the body, is the lowest level of parasites to be borne without clinical

symptoms; more bring on fever which continues till the parasites reach a low and tolerated level.

Case I.—L. A., Seamen's Hospital; double-tertian. The counts were as follows:—

| Date | Half-grown parasites | | Fully-grown parasites | | Fever |
|-----------|----------------------|---------------|-----------------------|----------------|-------|
| | In c.mm. | In body | In c.mm. | In body | |
| 1901 | | | | | |
| May 10 .. | 350 | 1,750,000,000 | 430 | 2,150,000,000 | + |
| " 11 .. | 70 | 350,000,000 | 280 | 1,400,000,000 | 0 |
| " 12 .. | Not seen | — | 100 | 500,000,000 | 0 |
| " 13 .. | 100 | 500,000,000 | 410 | 2,050,000,000* | 0 |
| " 14 .. | Not seen | — | 50 | 250,000,000 | 0 |
| " 15 .. | 700 | 3,500,000,000 | 410 | 2,050,000,000 | + |
| " 16 .. | 140 | 700,000,000 | 140 | 700,000,000 | + |
| " 17 .. | Not seen | — | 70 | 350,000,000 | 0 |

* Fever would have been found here if temperature had been taken often enough.

From the above figures and limit of two billions, it follows that the duration of initial latency depends upon the number of trans-stellated sporozoites.

Malignant tertian, sporulating every forty hours:—

| | | | | | |
|------------|------------|-----|---------------|---------|-----------|
| 1st day | 2 | 4 | 6 | 7 | 9 |
| 1 parasite | 20 | 400 | 8,000 | 160,000 | 3,200,000 |
| | 11 | | 12 | | |
| | 64,000,000 | | 1,280,000,000 | | |

= fever on 14th day, or 7th day if two sporozoites were injected.

Benign Tertian:—

| | | | | | |
|------------|------------|-----|---------------|---------|-----------|
| 1st day | 3 | 5 | 7 | 9 | 11 |
| 1 parasite | 20 | 400 | 8,000 | 160,000 | 3,200,000 |
| | 13 | | 15 | | |
| | 64,000,000 | | 1,280,000,000 | | |

= fever on 17th day, or on 8th day if two sporozoites were injected.

Quartan:—

| | | | | | |
|------------|-----------|------------|-------------|---------------|---------|
| 1st day | 4 | 7 | 10 | 13 | 16 |
| 1 parasite | 10 | 100 | 1,000 | 10,000 | 100,000 |
| | 19 | 22 | 25 | 28 | |
| | 1,000,000 | 10,000,000 | 100,000,000 | 1,000,000,000 | |

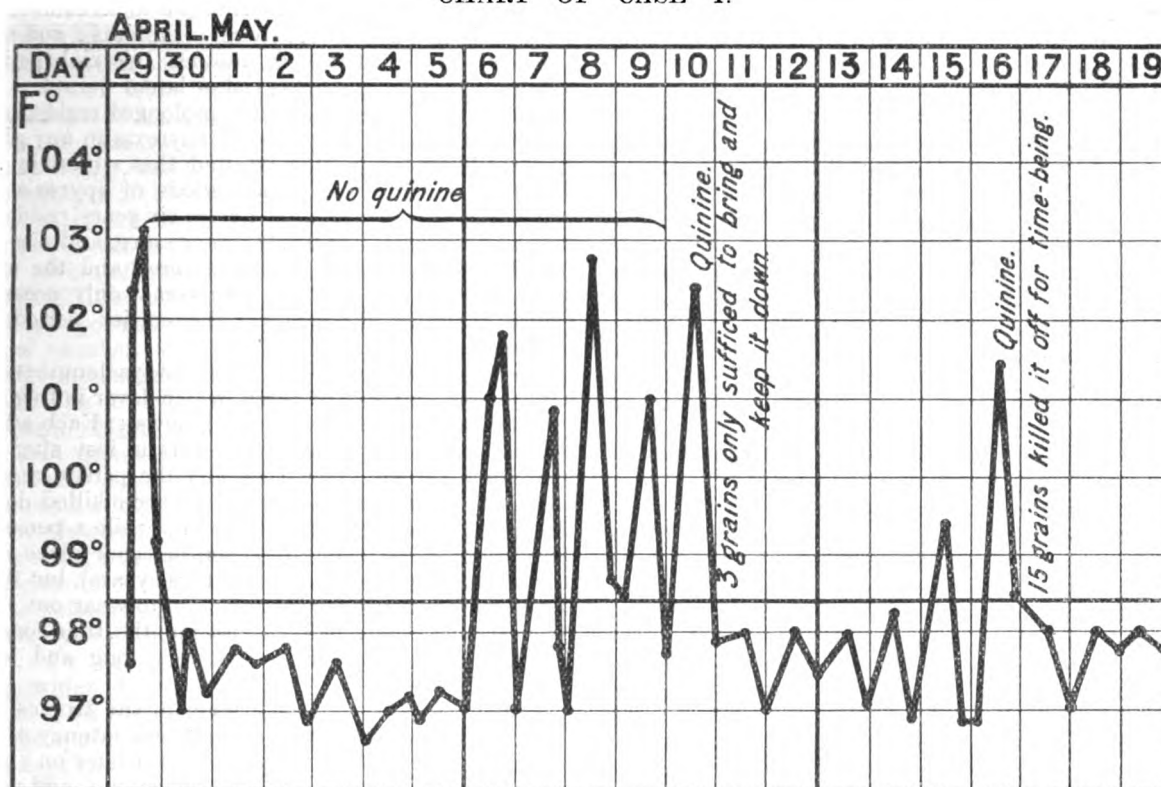
= fever on 31st, or on 15th or 16th day if two sporozoites were injected.

Ordinarily two only would account for the usually observed incubation period of five to fifteen days; were more injected it would be shorter, and if an "infantile mortality" were established of the sporocytes then it would be an indefinite period.

From the above calculations it is clear that, at this stage, malignant tertian has the shortest latency, next tertian, and quartan the longest. Lengthened variations of the primary latent period (incubation) have been observed. Some individuals have had fever for the first time on coming home. A student of the London Tropical School lived in Nigeria a year and had fever for the first time on sighting England; probably infection took place at the moment of departure. Cases are recorded of a long time before first symptoms appeared, though the parasites were there; possibly the initial fever, which occurred at the fixed time, was slight and unobserved. The primary latency is generally five to fifteen days, and nine months might elapse before a second attack (in England), as in the case of Dr. Manson, jun., and Mr. Warren, of the Tropical School; in another case it was well over a year before a second attack occurred.

While the foregoing is true initially, and explains incubation periods of the experiments upon Manson,

CHART OF CASE I.



Warren, &c., the subsequent periods of latency after a first fever cannot be fairly considered on the evidence of isolated prolonged intervals alone. It is necessary also, and perhaps mainly, to consider the ordinary conditions under which the parasite remains "latent," as far as clinical or symptomatic evidence of its presence is considered. This is the most important point to consider.

In cases untreated by quinine symptoms may increase, remain constant, or decrease. In such cases where blood examinations are made frequently it will be observed that similarly and correspondingly the parasites may become more numerous, remain in about the same numbers, or decrease in number. "During the first observed cycle there was an average of 500 parasites; during the second cycle an average of 446 per c.mm.; during the third an average of 627; and the fourth cycle, 302 per c.mm." (JOURNAL OF TROPICAL MEDICINE, p. 110, April 1st, 1901.)

History.—L. A., aged 28, seaman. His last voyage, February, 1901, was to Rosario, where he had several fevers. Was admitted to Seamen's Hospital, with fever, April 29th. Other than purges, rest and diet, he had no treatment at that time; the fever ceased the next day and he continued free for a week. Blood examination showed a double tertian infection and many parasites were seen daily. On being sent out for a walk the fever returned.

Appended is a chart of his fever, from which is seen that, after spontaneous decline of his fever, the parasites remained below fever level for a week. (See also case, JOURNAL OF TROPICAL MEDICINE, p. 178, June 1st, 1901, where the "special points of interest were:—(1) the absence of fever; (2) the

presence of well-marked tertian parasites in considerable numbers; and their disappearance without administration of quinine.")

If the parasites, as in the second case, or, as is common in a double infection, remain fairly constant at a low level no acute symptoms will manifest themselves, though organic changes, *e.g.*, splenic enlargement, may occur. It is an old observation that chronic splenic enlargement may, and often does, take place in persons who suffer little from acute attacks of fever. It is quite common also to find huge spleens in children of Europeans who have been brought up in the tropics, and yet such have had very little fever.

Case II.—M. P. Mc.I., Seamen's Hospital, May 23rd, 1901, aged 30, had two attacks of jungle fever in 1890, and three years after another attack. In India five years; then East Africa two years—no fever; then West Africa—had one attack of fever on board; was fifteen months in Lagos, and no fever; was six months in Borneo—had no fever there. Temperature normal. Spleen enlarged; extends almost to umbilicus and within two inches of the crest of the ilium. No malarial parasites.

This period of latency then is only one of clinical latency; the parasites are not "latent"; they are destroyed at the same rate, or approximately so, as they are produced; the "birth and death-rates" are about equal, the population sparse and consequently does not increase. Pushing the matter further, it is unlikely that parasites would be found in an ordinary examination if they were only present in the proportion of 1 in 1,000 leucocytes or 7 per c.mm. of blood.¹

¹ It is even hard to find them when only 35 to 40 per c.mm.

and yet this number would correspond in a person of average weight to :

Body weight of 65 Kilos.

$$\frac{13}{1000} \times 1,000,000 \text{ c.mm.} \times 7 \\ = 35,000 \text{ parasites.}$$

The question naturally arises whether this is the only manner in which "latency" occurs. The suggestion of a latent form of parasite has been frequently made, but no proof has been offered. The karyochromatophilic granules, or primitive forms of Plehn, "Plehn's bodies," are plainly degeneration forms of red discs; so evident is this that no one has thought it worth while to refute his theory about them. By some, at one time, the gamete form was considered to be a possible latent form. With our present knowledge of the sexual nature of the gamete this supposition can be excluded.

It has also been suggested that perhaps in bone marrow or elsewhere a few of the parasites may continue to sporulate and pass through the entire asexual cycle without appearing in the peripheral blood. This supposition does not in reality throw any further light on the question. It may occur, and the peculiar aching of the bones so common before malarial attacks perhaps supports the view; but the apparent absence of parasites in peripheral blood is just as easily explained on the supposition of the number present being small.

The next question that arises is the length of the period during which asexual reproduction of the parasites can be continued without fresh introduction of parasites invigorated by being sexually reproduced. Nuttall, of Cambridge University, has said of this: "Since the publication of Maupus' remarkable works we have become aware that, with all these protozoa, this faculty of non-sexual multiplication becomes exhausted in the long run, and the species would become extinct did not sexual reproduction intervene" (JOURNAL OF TROPICAL MEDICINE, 1900). Under favourable circumstances the period may be a prolonged one, but not interminable. The sum of the matter, in malarial cases of the tropics in England, is, that the parasites are latent in the body till they die down to extinction, because asexual reproduction is finite in about three years in quartan, two years in malignant tertian, and tertian in between these.

Clinically we have abundant evidence of repetition of asexual cycles for weeks (JOURNAL OF TROPICAL MEDICINE, pp. 178, 210, 1901; and the case L. A., previously given). In untreated cases, after a time the number of parasites falls (*cf.* case L. A., chart), but the fall may be slow and the decrease in number arrested by any change in the condition of the host, as exposure to cold, fatigue, excess, atony, illness, &c., and replaced by an increase.

Even without any such obvious condition there is a period when the parasites again become numerous, again diminish in number, and again increase. The observations actually reported on the number of parasites are too limited to be of much value, but of clinical evidence we have abundance (*cf.* case L. A., and others already referred to) that an attack of fever naturally subsides; that there is an interval of apyrexia, stated by Koch to be usually twelve days, but perhaps most commonly

from two to four weeks, and often up to three months, during which time there are no symptoms; and that after this period there is another pyrexial period, followed again by the apyrexial or latent period.

It is noticed also that with prolonged residence in a malarial country the period of apyrexia in any given case tends to become longer, and that whilst in the first two or three years the periods of apyrexia are two weeks or a month, after five or six years' residence they are often six months, or even more, and also the actual pyrexial attacks are less severe; and the most severe attacks of all, those with coma, only occur in highly malarial districts in the earlier periods of residence.

The explanation of the period (always lengthening) between two attacks of fever, which fever is frequent at first, then less frequent, is as follows: Each attack confers a temporary immunity; this is lost after the increasing periods mentioned, and the person regains immunity after the parasites have been killed down. There is no absolute limit to latency while a person is subject to reinfection (in a non-malarial place it is definite and terminable in about three years), but there is a strong tendency for the infection to wear out. The interval is variable; short in first months, then longer; and after a certain time it is very long and fever infrequent.

Thus in a newly arrived person in the tropics, and for the first time infected, there is less latency of the parasite, and little immunity from fever later on; after years there is greater latency of parasite, and much immunity from effects of the parasites and malarial fever, which is only lit up at longer intervals. Still later in life there may be no parasites in the blood and immunity is reached, which some people do acquire in the Roman Campagna in spite of reinfection, which is harmless to them. This is seen also in bird malaria, where the old birds get immunity like man.

All this indicates an adaptation of the person to his parasites so that a limited number only will be tolerated, but that these up to a low limit will not give rise to sufficient reaction to cause their complete destruction.

It will be seen from the above that it is conceivable that the period of latency is a very indefinite one. It is only terminated by the complete destruction of the parasites.

The causes that lead to their destruction are unknown, but seem to be more active with numerous parasites, and diminish in activity when the parasites become very few, so that complete destructive agencies are maintained with parasites at a low level.

The clinical evidence of duration is in accord with the above. Recurrences of fever when removed from all possible sources of infection are very common for three months; are still common up to six months; but become rarer after that period. Up to a year, however, they cannot be considered as exceptional. In illustration one may quote the cases of Manson and Warren. Dr. Daniels had fever after twelve months' interval from the previous one, or ten months counting from last possible reinfection. It is two years since my last fever in Zanzibar. I have had three fevers during the three years of my absence from the Congo.

There is a large amount of negative evidence of recurrence of fever over two years.

Certain favourable and most important deductions may be drawn from latency when free from possible reinfection. If a person in England has had no fever during the first two months of return it is a very favourable sign, and if five months have elapsed then the possibility of bilious hæmaturic fever is remote.

The existence of crescents may be reckoned in weeks, after which they disappear.

The action of quinine upon latency is remarkable. A suitable dose kills off about 99 per cent. of the spores at sporulation, *i.e.*, reduces them to $\frac{1}{100}$ th of original number, after which the body, probably by the large mononuclear leucocytes, keeps them at a low level till the fever is relit or continues the progress to extinction. More probably, and more reasonably expressed, quinine *helps* the large mononuclears to assert their acquired inhibitory power to keep the parasites at a low level tolerated by the body. This is done by phagocytosis. If a dose of quinine kills off 99 per cent., a larger dose might be expected to kill them quite off; generally it does not, for while it is a specific poison to the spores, it very seldom kills them off entirely. A certain number remain unkilld and latent. Dr. Manson thinks that it acts upon something, which in its turn acts upon the parasites; quinine *helps* to fight the parasites.

If the spontaneous cure of malarial fever and their being kept low and comparatively harmless is effected by the large mononuclears, it may find a possible elucidation in the analogous process observed in relapsing fever (*Pasteur Institute Annales*, August, 1901). Here the natural cure is brought about through the phagocytic action of the polymorphonuclear leucocytes. These kill off the spirochetes when the fever is highest by a corresponding and simultaneous great increase of themselves (18,000 leucocytes to c.mm.) As in malarial fever the large mononuclears only are increased from the normal 8 per cent. to 30, 50, or 60 per cent., and the polymorphonuclears diminished, probably here we may find the *rationale* of the parasite's disappearance by phagocytic action of the large mononuclears. This action is often ineffective to utterly destroy them, with or without quinine, resulting in a latency of the parasite.

Quinine then, taken in fever or prophylactically, will *help* to keep the parasites at a low level, and therefore comparatively harmless. Plehn in his "Weiteres über Malaria," maintains that at Cameroons, by giving half a gramme every five days he has increased the apyrexial periods, or latency, to about twelve to fourteen months on an average; increased the possible length of service of officials, and lessened the frequency, intensity and death-rate of fever; and furthermore diminished in consequence the tendency to black-water fever.

One-third of a grain given thrice daily in malarial fever of a suitable subject will keep the parasites at a low level. On the other hand I have seen cases where one drachm a day did not reduce the parasites or save the patients. Probably in such cases there was an unhealthy phagocytosis. A great increase of immature ineffective large mononuclears, having no phagocytic action, returning home empty as it were, would

account for the ineffectiveness of quinine in these cases.

That the use of quinine tends to increase the duration of latency or apyrexial periods, keeping the parasites low, should be most important in practice (*JOURNAL OF THE TROPICAL MEDICINE*, p. 179, June, 1901), and completely rectify the idea of only giving the drug to meet and kill off the spores at the moment of their liberation during the fever.

Case III.—H. R., in Congolese Africa more than twenty years. Habit, through all these years 3 to 5 grains quinine daily prophylactically; had good health while so doing and never bilious hæmaturic fever. Returned to the Congo in 1901 after two years at home. Ceased the preventive use of quinine with the idea that the drug was only effective at moment of parasite's sporulation. Had his first attack of black-water fever almost immediately. Now returned to old habit.

PETANELLE.

A PREPARATION OF FIBROUS PEAT.

MESSRS. PATÉ, BURKE AND Co., 6, Wool Exchange, London, E.C., have favoured us with specimens of their preparations. In many ways these preparations appeal to us from the standpoint of the wants of residents and practitioners in the Tropics, and we desire to draw special attention to this material in its several phases and applications.

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Current Literature.

TROPICAL ULCER AND ITS TREATMENT BY HOT WATER.—Roux speaks of an ulcer common to the natives of the west coast of Madagascar, and its treatment by hot water. It does not often attack Europeans, since their conditions of life are so different from those of the natives. It is found chiefly in the labourers—those subject to wounds and infections. It frequently has its origin in an injury caused by small thorns which are produced by certain kinds of native herbs. These penetrate the skin, and an abscess eventually forms and becomes the seat of various infections. This ulcer is very frequent in its occurrence and shows great resistance to the ordinary remedies. Excellent success has attended the use of hot water of 55° to 60° C. (131° to 140° F.). The water is boiled for fully

fifteen minutes, and is then cooled down to about 55° C. The water is applied first by squeezing it out of a pad on to the ulcer, care being taken to protect the neighbouring parts. A pad soaked in the water is next applied and allowed to remain some minutes on the ulcer. Next a compress of gauze wet in the hot water is laid on the ulcer previously covered with vaseline; over this a pad of absorbent cotton is fixed by a bandage. The dressing is renewed every day and the limb is kept at rest. The cure is speedy; the suppuration ceases, and cicatrisation is rapid. When the cure proceeds more slowly, and the presence of syphilis is suspected, iodide of potassium is administered. Ulcers 7 cm. in diameter have been healed in three or four weeks. The writer states that he has now employed this treatment for two years and has discarded all other methods. It is probable that the hot water acts directly on the micro-organisms in the wound, attenuating their virulence, and on the general organism, increasing its resistance to the action of the microbes. As soon as the application is made, the base of the ulcer reddens, showing a reflex vasodilator action very favourable to phagocytosis. The cells themselves are reflexly excited, their vitality is increased, and they multiply.—*Lc Caducée*.

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The Journal of Tropical Medicine.

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Original Communications.

THE PART PLAYED BY THE FLEAS OF RATS AND MICE IN THE TRANSMISSION OF BUBONIC PLAGUE.

A Critical Note.

By Dr. BRUNO GALLI-VALERIO.

Professor at the University of Lausanne, Switzerland.

(Translated by P. Falcke.)

THE part played by certain insects and arachnides in the transmission of various parasitic diseases of man and beast is well known. It will suffice to recall the important rôle the mosquito plays in the transmission of the malaria of human beings and birds, of yellow fever, and of the filariasis of men and dogs; the rôle of the Tsé-tsé fly in the transmission of naganà; of ticks in the transmission of malaria of cattle and dogs; and the part played by flies and other dipteræ in the transmission of anthrax, &c.

These facts probably induced Dr. Simond¹ to open up the question of the transmission of bubonic plague to man from rats and mice through the instrumentality of the fleas of these animals.

Ogata² had previously, in 1897, conveyed plague to mice by inoculating them with triturated fleas collected from plague-infected rats. M. Simond confirmed the presence of a bacillus, morphologically similar to the plague bacillus, in the fleas taken from rats suffering from plague. Of three mice, inoculated with these fleas pounded up in a few drops of water, one only succumbed to bubonic plague.

M. Simond, besides, observed the death of one rat and one mouse placed in a cage with a rat that had died of plague, to which he had previously added fleas from a cat (!); whereas of seven rats placed in a cage

with a rat that had died of plague, but which exhibited no fleas, none of the seven showed symptoms of plague.

In consequence of these observations, M. Simond's attention was arrested by initial bullæ observed on persons suffering from plague (one case in every twenty), and in which he found the plague bacillus.

According to M. Simond these blebs were due to the bites of fleas of rats and mice. The mode of infection, according to M. Simond, is supposed to be as follows: "The sting (*sic*!) steeped with blood cannot for long maintain its power of infection. The flea in these cases would be useless except at the moment when it leaves the plague-stricken animal. But it is feasible to suppose that during suction the flea deposits its dejecta, consisting of a tiny drop of digested blood, on the place where it is perched. In cases where this fluid is a culture of plague bacilli it is feasible to conjecture that it may infect the person through the perforation made by the sting."

From these facts M. Simond draws the following conclusions: "While recognising that this hypothesis has not the value of a demonstrated fact, we are of belief that the various forms of 'spontaneous' plague in men and animals generally take place by one single mode of infection—intracutaneous parasitic inoculation."

M. Simond's work made some sensation, but instead of being enquired into, his views were accepted as they stood, and above all by the French, who had no hesitation in affirming that the transmission of plague from rats and mice to man took place through the intermediation of fleas, and that this was the most important means of transmission of the disease. Before attacking M. Simond's theory, we will first consider what publications have appeared subsequently, having for their object the support of the theory of the transmission of plague by fleas.

There is, first of all, an article by M. Loir, Director of the Pasteur Institute at Tunis.³ He commences as

¹ *Annales Pasteur*, 1898, No. 10, p. 626.

² *Centralbl. für Bakt.*, 1897, xxi., Nos. 20, 21, p. 769.

³ *Revue Scientifique*, 1900, No. 13, March, p. 895

follows: "It has now been demonstrated that the flea is the principal intermediary of the plague between the rat and man."

A curious affirmation, when M. Simond himself had declared that his theory had not the value of a demonstrated fact! Starting with such premises it is easy to arrive at conclusions favourable to M. Simond's theory.

In discussing this affirmation M. Loir explains why a certain immunity as regards plague is noticed in persons working with oil. The presence of oil on the skin drives the fleas away. This is how M. Loir and his assistants made their experiments: "We shut up a rat in a cage and afterwards placed it for twenty-four hours in a house in Tunis in which we knew by experience that fleas swarmed, inasmuch as, during a medical visit to a street porter living in this house, I had removed thirty-seven fleas from my garments in the evening. At the end of twenty-four hours the rat was swarming with these insects. We placed two small cages within this cage, one containing an ordinary rat, the other a rat previously dipped in olive oil; we then killed the first rat and left its body in the large cage at an equal distance from the two small cages. On the following day, of course, not a single flea was to be seen on the dead body. The rat that had been placed in the first of the small cages was covered with fleas, but the rat dipped in olive oil had not a single one." M. Loir concludes from this experiment that it is perhaps this distaste of the rats for oil to which the immunity of all workers in oil-factories is due, a circumstance that has been remarked in all epidemics of plague.

Another work supporting M. Simond's views is by Dr. Ashburton Thompson,¹ Government medical officer at Sydney. During the epidemic in this city Dr. Thompson observed the blebs described by Simond in six cases; a small proportion, he observes, in relation to the great number of cases examined. In one of these pustules he found a bacillus, similar to *B. pestis*, in small numbers. Dr. Thompson then examined fleas taken from plague rats, and in one flea he found *B. pestis*, which proved destructive to guinea pigs. Of nine specimens of fleas he found, on the rats he had examined to find out the species, two were *P. serraticeps* and the others *P. fasciatus*. In consequence of these facts Dr. Thompson thinks he is entitled to conclude that the transmission of plague from rats and mice by the intermediation of fleas must be very frequent.

During the same epidemic Dr. Tidswell,² Bacteriologist to the city of Sydney, also pronounced favourably in support of M. Simond's theory. He gave plague to rats by inoculating triturated fleas taken from plague rats; and he observed that at the time the quays of the city were covered by dead rats, that the fleas swarmed into the streets to such a degree that the labourers were obliged to tie the bottom of their trousers with string round their feet, in order to protect themselves from the onslaughts of these vermin.

Let us now undertake a critical study of these articles.

Dr. Nuttall³ is the first to throw doubts on M. Simond's affirmations, for he observed that the bacteria passing through the body of fleas and other insects often succumbed. In conclusion he observes: "What we want in this respect is more facts and fewer opinions, and the facts can only be gathered by further experimental research."

Without having any knowledge of the work of Dr. Nuttall, which the author only sent to me after my article had been published, I also submitted a study which was critical and, to a certain degree, experimental on M. Simond's theory,⁴ and it is to a great degree the contents of these investigations which, in addition to the new facts and the new criticisms, I shall give here.

In the first place the initial blebs, of which M. Simond has spoken as a point of entry by the bites of fleas, has been observed by him in too restricted a number to convince us that this mode of infection is the most frequent.

This rarity forcibly struck Dr. Thompson, who notes how small the proportion of cases of blebs are in comparison to the cases examined, and of the six cases examined only one contained *B. pestis*. It is true that M. Simond, to explain the affair, evolves the theory that if the flea inoculates a very active virus the bleb will not be produced, though the infection is conveyed. But as M. Simond admits that the infection does not take place by direct inoculation, but by means of the bacilli passed through the digestive tract of the fleas, and as, according to the experience of Nuttall, these bacilli in most cases are attenuated, if not dead, it appears to me that if the indicated mode of infection were frequent blebs also should be much more common.

M. Simond, in expounding his theory, has taken no trouble to find out if the fleas that live on rats and mice are of the same species as those that live on man. This is what he says: "The flea commonly met with on the wall rat (India) is of medium size, of a greyish colour, with a spot the colour of dregs of wine on the lateral aspect of the abdomen; this spot is nothing but the stomach filled with blood seen in consequence of transparency."

"We are not aware if this flea is a different variety from the ordinary fleas of men and domestic animals (*sic*). At the same time we have convinced ourselves experimentally that, transmitted to persons or dogs, they immediately attack them."

Such a confusion of the forty-eight species of common fleas is not adapted to inspire great confidence in the author's experience. Indeed, how can we positively assert that the flea in question immediately bites man and the dog if we do not even understand what flea is in question; and a little time after, to make confusion more confounded, M. Simond records experiments made with fleas taken from the cat.

A deplorable analogous confusion is exhibited in

¹ *Journal of Hygiene*, vol. i., 1901, p. 153.

² *Journal of the Sanitary Institute*, 1901, p. 509-578; *Résumé des la Rev. d'Hyg.*, 1901, p. 553.

³ "On the Role of Insects, Arachnides and Myriapods as Carriers in the Spread of Bacterial and Parasitic Diseases of Man and Animals." *Johns Hopkins Reports*, vol. viii., 1899.

⁴ *Centralbl. für Bakt.*, 1900, xxvii., No. 1, p. 1, and xxviii., No. 4, p. 842.

M. Loir's work, who classes all the fleas together and tries to show that there is only one species. The first question to settle is the zoology of the fleas, and then discuss the question whether the specimens found on rats and mice can attack man.

I set to work to ascertain the nature of these parasites and find that the fleas met with on rats and mice under normal circumstances are *Typhlopsylla musculi* and *Pulex fasciatus*. The first mention made in literature, and this after the appearance of my work, is by Dr. Thompson, who asserts that among fleas collected on rats there were two from which he obtained *P. serraticeps*. This specimen is a flea of the carnivora and is very frequent on dogs and cats. It sometimes passes on to man, and Railliet as well as myself have also found it on the rabbit.

In order to confirm which species besides *Pulex irritans* bite man, this being man's particular flea, I made some experiments on myself, and when permitted on other persons. Here are the results:—

Typhlopsylla musculi.—In one case in which I was invaded by numerous fleas of this species coming from a white mouse I did not receive a single bite and all the fleas quickly left me. Some of these fleas, placed expressly on my body and left free or under glass bells, never bit me although fasting.

Pulex fasciatus.—What I have reported of *T. musculi* stands good for this species.

P. serraticeps.—This species, as is known to numerous observers, bites man. I have myself found it on human beings. Placed on my body under a glass bell it immediately bit me.

P. gonioccephalus.—This specimen, conveyed from the rabbit to my body, did not bite me.

P. avium.—A variety which, according to M. Lucet may bite man, being placed on the body of a colleague and myself caused no wound although it had been kept fasting a long time. I may remark that the specimen employed for this experiment emanated from a *Chelidon urbica*.

P. erinacei.—I was invaded by numerous fleas of this species. I placed some hundreds under a glass bell on to my body. Although I had not felt any bites from those that had been free on my body, yet those under the glass bell bit me slightly. They remained only a short time on my body.

The experiments quoted are not of absolute value as they were mostly made on myself and not on several persons, but their value is increased by the fact that I was bitten by different species, such as *P. irritans*, *P. serraticeps* and *P. erinacei*.

From these experiments it appears that *P. fasciatus* and *T. musculi* do not seem to bite man. There remains *P. serraticeps*, which Thompson affirms he twice gathered off rats. But *P. serraticeps* must be very uncommon on rats. I have examined numerous rats without ever finding a specimen. Nobody, with the exception of Dr. Thompson, has quoted it as being found on this animal. On rabbits, where it was found by M. Railliet and myself, it is scarce, and M. Railliet's efforts to acclimatise this species on a rabbit by introducing a number into the hutch were unsuccessful. Dr. Thompson's observation therefore palpably relates to a rare case, an accidental case, and we cannot rely on this observa-

tion for the purpose of considering this flea as the ordinary medium for transmitting plague from rats to man.

Dr. Tidswell, as M. Vallin admits, shows that he has a certain partiality for this almost exclusive mode of infection by means of the bites of fleas, gives absolutely no facts to support his theory. In fact he failed to give plague to rats placed in cages separated by large-holed lattice work from other cages in which there were plague-stricken rats, or even by placing healthy rats in dirty cages where other rats had died from plague. Quite recently Kolle¹ has also asserted that he could not succeed in giving plague to healthy rats placed in cages containing numerous fleas from off plague-infected rats, although he could positively affirm the fact that these fleas went on to the rats in the cages. He concludes that if the transmission of plague from rat to rat by means of flea-bites exists it has not been proved.

We know that Simond only had the death of one rat and one mouse placed in a cage with fleas from a cat originally taken from a plague rat. If transmission is so difficult from rat to rat, why, on the other hand, should it be so frequent from rats and mice to man, who is not as a general rule attacked by mouse and rat fleas.

Dr. Tidswell tells us that the abundant fleas on the quays at Sydney, probably from dead rats, attacked men's legs. But these fleas were not examined to ascertain if they were mouse and rat fleas; we are not informed if they bit the men or not, and we are not told if these men, invaded in this fashion, were more attacked by plague than others. Concerning these matters M. Vallin, though in favour of M. Simond's theory, writes: "At the same time Dr. Tidswell has not furnished us with sufficient details concerning his experiences, and has not supported the numerous assertions in his work by proofs."

To all this I must add that neither the German Commission nor Mr. Schotelius² in India, nor during the epidemics at Oporto, Glasgow, or Naples, has the transmission of plague to man been traced to the fleas of rats and mice. In opposition also to the assertion that this mode of transmission is the most frequent and almost exclusive (even taking into account the great facility with which plague is transmitted to man by the *Arctomys bobac*, on which, as far as I know, there are no fleas), is the fact noted by Edington,³ that at the Cape the rats that died of plague did not succumb to bubonic plague, but to a disease due to a bacillus analagous to *B. pestis*, which is non-pathogenic to guinea-pigs and pathogenic to pigeons.

The facility also with which it is possible to arrest an epidemic of plague where hygienic conditions are good, and isolation properly carried out, speaks against this theory.

If M. Simond's hypothesis were correct, one might almost fold one's arms in consequence of the difficulty of preventing the diffusion of infected fleas, which, according to Tidswell, were met with in millions where rats had died of plague.

¹ Zeitschrift f. Hyg. u. infect., xxxvi., p. 397.

² Hyg. Rundsch., 1900.

³ Centralbl. für. Bakt., xxix., No. 23, 1901, p. 889.

In conclusion, instead of asserting—as is too often the case—as an established fact that plague is transmitted to man by the bites of the fleas of rats and mice, and that this is the most frequent and important means of infection, it requires to be demonstrated, not only that the fleas pass from rats and mice to man, but from rat to rat.

The question can only be solved in one way, namely, by conveying to the bodies of human beings rats' and mice's fleas that have lived on plague rats. If this experiment is, as I believe, considered to be necessary, I place myself entirely at the disposal of the committee to undergo it.

ADDITIONAL NOTES ON MALARIAL FEVER IN ST. LUCIA; AN ANALYSIS OF 230 CASES.

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A FEW months ago Dr. G. C. Low and I sent a short note to the *British Medical Journal*,¹ on the prevalence of malaria in St. Lucia.

Since Dr. Low left St. Lucia to continue his researches in Barbados, British Guiana and St. Vincent, I have followed up the subject on similar lines. In all I have collected notes of some 230 cases of malarial infection representing about 400 blood examinations of patients selected from 2,433, who came to the Castries Dispensary with various complaints for medical advice and treatment. As I was working alone, and without Dr. Low's valuable assistance, and as the time at my disposal was limited, I was obliged to make a still more careful selection of cases for blood examination than in the first series. Only those who gave a definite history of fever, or who had febrile symptoms, or in whom I had reason to suspect malarial infection, were examined. No children who did not present symptoms of fever were examined for malarial parasites.

Most of the slides were examined wet as in the first series, but a certain proportion of dried and stained films were examined also, the stains used being borax-methylene blue or eosin and hæmatoxylin. The latter stain is especially useful when it is necessary to count the leucocytes in cases of suspected malarial fever where no parasites can be found. In these cases examination should be made both of wet films and of dried and stained preparations of the blood.

I can confirm the observations of Drs. Stephens and Christophers as to the increase in the number of large mononucleated leucocytes being diagnostic of malarial fever. I consider this one of the most important points elicited by those at present engaged in the investigation of malaria. These leucocytes, the "large hyaline cells" of Kanthack and Sherrington, are the largest of all the leucocytes, and are easily recognised, being usually ovoid or irregularly spherical in shape with clear, non-granular protoplasm. They undergo active amœboid movements and are phagocytic. They are the leucocytes which become pigmented in malarial

fever. The only other diseases in which the proportion of large mononucleated or hyaline leucocytes is increased, are lymphosarcoma, the cachexia of malignant disease, and in terminal leucocytosis, and then only occasionally. In every case in which I have found malarial parasites and have made a numerical estimate of the different varieties of leucocytes this increase has been observed. Here are a couple of examples taken at random from my note book.

CASE 229.—T. B., female, age 25, complains of fever every third day; five months pregnant. Blood examination shows quartan parasites and rosettes.

| | | | |
|------------------------------|---|-----------------|-------|
| Large mononuclear leucocytes | { | Hyaline .. | 19.37 |
| | | Transitional .. | 12.11 |
| Polynuclear | { | Neutrophile .. | 41.89 |
| | | Eosinophile .. | 1.21 |
| Lymphocytes.. .. . | | | 25.42 |
| 100.00 | | | |

CASE 383.—F. B., female, age 18. Pyrexia: indefinite symptoms. Blood examination shows malignant rings, pigmented leucocytes and crescents.

| | | | |
|------------------------------|---|-----------------|-------|
| Large mononuclear leucocytes | { | Hyaline .. | 28.68 |
| | | Transitional .. | 8.45 |
| Polynuclear | { | Neutrophile .. | 37.87 |
| | | Eosinophile .. | 1.10 |
| Lymphocytes.. .. . | | | 23.90 |
| 100.00 | | | |

Normal blood examined at the same time and stained in the same manner.

| | | | |
|--------------------------------|---|-----------------|-------|
| Large mononucleated leucocytes | { | Hyaline .. | 4.47 |
| | | Transitional .. | 3.89 |
| Polynuclear | { | Neutrophile .. | 64.40 |
| | | Eosinophile .. | 1.95 |
| Lymphocytes | | | 25.29 |
| 100.00 | | | |

I have had no opportunity of examining the blood in yellow fever but if it should turn out that there is no such increase of the large mononuclear leucocytes, then we have a valuable means of differential diagnosis between these two diseases, especially where quinine has been administered and so interfered with the examination for malarial parasites.

The transitional forms are large mononucleated leucocytes intermediate between the hyaline and polynuclear leucocytes, with indented or kidney-shaped nuclei, which stain more deeply than the nuclei of the hyaline cells. They resemble sometimes the hyaline and sometimes the polynuclear cells, while they vary in size, so that it is often extremely difficult to decide whether some leucocytes should be classified with the small or large mononuclear forms. In the above examples all doubtful forms have been classified as transitional, and only hyaline cells, about which there could be no doubt, have been included under that head.

THE PARASITES.

In 171, or 75 per cent. of the infected cases, malignant parasites were found. Thirty-six of these cases, 21 per cent. of the malignant infections, or about 16 per cent. of all the infected cases, had crescents in the blood, and were therefore sources of danger to their neighbours.

The most dangerous age appears to be the time of

¹ Published January 25th, 1902.

puberty and adolescence, for not only was the greatest proportion of malignant infections found among those from 10 to 20 years of age, but also the greatest number of crescent cases, and the most severe types of fever were met with in children near the age of puberty and in young adults.

The malignant parasites met with are the ordinary unpigmented ring forms. Pigmented rings are not uncommon, but they are always accompanied by unpigmented forms. I have never seen them alone, and consider the pigmented malignant rings to be merely a further development of the ordinary unpigmented rings.

I cannot say that I have seen the unpigmented quotidian parasites, or *Hæmaphysa immaculata* of Grassi and Feletti, although I have seen minute rings in a few severe cases which I believe were only young parasites.

In the course of my investigations I have been able to trace certain phases in the development of the crescent. One thing appears to be certain—it is always intracorpuscular while in the circulation; and in my opinion it only becomes extracorpuscular when it exflagellates and the flagellæ burst through the limiting membrane of the corpuscle in the case of the microgametocytes, or, in the case of the macrogametes, when the small globular bodies (polar bodies) make their appearance on the periphery of the non-flagellating spheres.

When a parasite (A) is about to become a crescent, instead of breaking up into spores its protoplasm appears to become encapsuled within the corpuscle. The pigment collects in the centre of the parasite, which has now become spindle-shaped (B). As the young crescent grows the ends of the spindle touch the limiting membrane of the corpuscle (C), and as it is prevented by this limiting membrane from growing longer as a spindle, it is bent in the form of a crescent (D). Finally, the pressure of the two ends of the imprisoned spindle (which is very elastic) on the limiting membrane of the corpuscle causes it to stretch and give way, but not to rupture. The ends of the crescent now appear outside the circumference of the corpuscle (E), still covered, however, by a thinner portion of its capsule—just as when one tries to push the end of the finger through a thin sheet of india-rubber which has been put on the stretch. The capsule of the corpuscle has not, however, been completely overcome, for it is still sufficiently strong to prevent the crescent (F) from straightening out into a spindle again until it leaves the circulation and becomes subject to external influences.

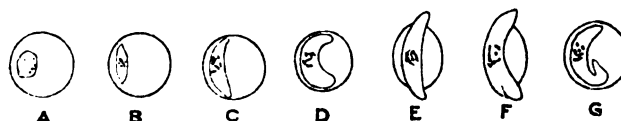
Sometimes the crescent is unable to push its way through the capsule of the corpuscle and curls up within it (G). These cases are rare. I have seen only one, and in this the patient had taken quinine three days before. Here it is possible that the quinine may have had some inhibiting influence on the growth of the crescent, the result being a degenerate, or rather immature form.

I see no reason for adopting the suggestion of Mannaberg, viz.: that the crescent is the result of the conjugation of two ordinary parasites in a doubly infected blood corpuscle. It would be just as reasonable to suppose that the tertian and quartan gametes

are derived from the conjugation of two tertian or quartan parasites in the same corpuscle.

Benign tertian and quartan infections are comparatively rare and do not call for any particular comment. Quartan infections are seldom met with and are the rarest of all forms of malarial fever seen in this colony.

Double infections, i.e., benign tertian and malignant parasites in the same person, were found in eight cases, or about 3.5 per cent. of the whole. In one of these both the benign tertian and malignant parasites were sporulating at the same time and I had an opportunity of comparing the two forms of "rosettes" in the same field of the microscope.



A, Malignant parasite; B, the same, having become spindle-shaped; C and D, young crescents within the corpuscle; E, the ends of the crescent pushing through the limiting membrane of the corpuscle; F, the crescent body as usually seen; G, crescent curled up within the corpuscle (from a case that had had quinine three days before).

Pigmented leucocytes alone were found in twenty cases, or 10.87 per cent. This large proportion is, no doubt, due to the fact that I was sometimes pressed for time, and made a hurried diagnosis of malarial fever as soon as I had found evidence of infection—either pigmented leucocytes or some form or other of the malarial parasite—without making a complete examination of the slide. A more prolonged search in a number of these cases would probably have resulted in finding parasites as well.

| | AGES | | | | TOTAL |
|-----------------------------|--------------|---------------|---------------|---------------|----------------|
| | Under 2 | 2—9 | 10—20 | 21 and over | |
| Malignant parasites .. | 14 50 % | 44 57.89 % | 44 63.78 % | 33 57.90 % | 135 58.69 % |
| Crescents .. | 5 17.86 % | 10 13.16 % | 13 18.84 % | 8 14.03 % | 36 15.65 % |
| Benign tertian parasites .. | 2 7.14 % | 8 10.53 % | 4 5.80 % | 6 10.53 % | 20 8.70 % |
| Quartan parasites .. | 0 .. | 4 5.26 % | 1 1.45 % | 1 1.75 % | 6 2.61 % |
| Double infections .. | 2 7.14 % | 2 2.63 % | 2 2.90 % | 2 3.51 % | 8 3.48 % |
| Pigmented leucocytes .. | 5 17.86 % | 8 10.53 % | 5 7.23 % | 7 12.28 % | 25 10.87 % |
| Total infected .. | 28 | 76 | 69 | 57 | 230 |

The pigmented leucocytes are easily recognised as large ovoid or irregularly-shaped cells, the clear protoplasm of which is dotted with coarse or fine granules of intensely black pigment. These granules, in doubtful cases, may perhaps serve as a guide to the nature of the infection. I have observed that when the granules are coarse and large the parasite, when found, is malignant, and that when they are fine the parasite is of the benign tertian or quartan type. This may be only a coincidence and needs further investigation before it can be accepted as an estab-

lished fact. The number of mild infections seen here is so small that observations would have to be continued for many months before this could be confirmed or refuted.

The severity of the symptoms has no apparent relation to the number or nature of the parasites found in the peripheral blood. I have seen a patient extremely anæmic, with high temperature, and apparently very ill, but could find only quartan parasites in the blood. This case was complicated by intestinal worms, which were probably the chief cause of the anæmia. But in other cases in which the patients were really very ill indeed (one or two were moribund) it was only after a prolonged search that I was able to discover perhaps one or two malignant rings or a pigmented leucocyte. I do not refer to cases in which the patient has had quinine, for then it is always difficult, often impossible, to find parasites at all. On the other hand, I have seen the blood of patients who complained of nothing more than a slight feeling of malaise, swarming with malignant parasites, or even with crescents. One woman who came to me said that she had not had fever for more than a week and that she felt quite well but wanted some medicine to give her strength. When I examined her blood with the microscope, however, I found from one to three crescents in nearly every field. Such cases are of course unusual, and when numerous parasites are found in the blood the patient is generally fairly ill.

I do not find very young children more susceptible to malarial fever than others, for out of a total of over fifty children under 2 years of age, all of whom presented some symptoms of fever, only twenty-eight, or about half, were infected. I have not yet been able to find the parasite in children who had not some febrile symptoms.

Natives of the place do appear to acquire some degree of immunity as age advances; but this immunity is not very great, nor is it constant, for I have seen very severe infections with crescents in old people over 60 years of age. Strangers from other islands, especially from Barbados and Montserrat, appear to be more susceptible to the malarial infection than natives of St. Lucia. Dr. Low has been unable to find Anopheles in Barbados, where all the cases of malaria are, as far as I know, imported.

This second series of cases bears out what we stated in our former paper, that in St. Lucia the most unhealthy season of the year for malaria is during the months of January, February, and March. In the first series of cases, out of 1,061 people seen between January 14th and April 4th, suffering from all kinds of diseases and injuries, 137, or 12.91 per cent., were malarial infections. In the second series, embracing the period between the middle of April and the end of August, 2,433 people were seen and only 230, or 9.45 per cent., were infected. I do not believe that in either series we missed a dozen cases of malarial infection from neglecting to examine blood films. It is to be regretted that these cases could not be followed to their termination, but as most of the patients came to the dispensary once only, seldom twice or oftener, and then disappeared, this was impossible.

HINTS ON THE PREPARATION AND EXAMINATION OF BLOOD-FILMS FOR MALARIAL PARASITES.

I generally see my patients at the dispensary with my microscope in a convenient position on the table beside me. Close at hand are a few pieces of old cloth about six inches square, a few glass slides and cover-glasses, a needle or other instrument for puncturing the skin (I use a hare-lip pin with the point ground very sharp), a small, wide-mouthed bottle of spirit, and a small basin of water. The latter should be placed as far away from the microscope as possible. When a patient comes in whom I think it necessary to make an examination of the blood, I clean a slide and cover-glass with a piece of the old cloth and a drop of spirit. Then I sterilise the needle and clean the patient's finger-tip or the lobe of the ear with spirit and make the necessary puncture. I have observed that the smaller the droplet of blood taken up on the cover-glass the better the film. It is a mistake to take too large a drop. It is better, and saves time in the end, to wipe away the blood over and over again until one gets a droplet of the proper size. I do not ring the preparation with vaseline, as a rule, unless I wish to make a more extensive study of it. When a convenient field is found with the low power objective, the nose-piece is revolved and the highest power used. If one has no revolving nose-piece a proper field can be quickly found by focussing the extreme edge of the film with the high power objective, and then drawing the slide slowly across the stage in such a manner that the centre of the film passes under the objective. In this way the different tones of the film come into view and can be examined. Care must be taken to focus the *extreme edge of the film*, for the zone of free hæmoglobin is often perfectly homogeneous, and should one attempt to focus that, the high power objective may be brought down upon the cover-glass if one is not very careful.

If parasites are not seen in the first two or three fields I usually go rapidly over the whole slide, looking out for pigmented leucocytes or crescents. These are readily recognised by their large size and black pigment, and, in the case of crescents, by their very definite shape. Of course, if a parasite should appear in the field I stop to identify it and make any observations that may be necessary. The amoeboid movements of the malignant parasites in freshly drawn blood are so very active that it is often impossible to see them until they become quiescent. By the time one has gone over the whole slide and determined the presence or absence of pigmented leucocytes or crescents, these movements will have slowed down and the parasites, if present, will have become visible. Malarial parasites have a higher specific gravity than hæmoglobin, and, when at rest, will be found by focussing the *lower surface* of the corpuscles in which they are.

When the examination of the slide is finished I drop it into the basin of water and the nurse cleans it and the cover-glass, while I make the necessary entry in my note-book and prescribe for the patient.

The usual fallacies of mistaking vacuoles for parasites, dirt for pigment, &c., can best be avoided by knowing and recognising every object that appears in

the field of the microscope. Bacteria are nearly always actively motile, and are usually too small to be mistaken for malarial parasites. Sometimes a blood-plate will rest upon a corpuscle and simulate a parasite, but it does not exhibit amœboid movements, it is nearly always crenated, and can be made to move away from the corpuscle by tapping on the cover-glass with a needle. This tapping on the cover-glass is of use also as a means of making a crescent come out of its hiding-place when in the zone of heaped-up corpuscles. Occasionally something black is seen among the corpuscles, but these are so close together that it is difficult to see just what it is until the corpuscles are set in motion, and the black object is then seen to be the central pigment mass of a crescent.

Films that are to be dried and stained can be most conveniently and rapidly made by spreading out a drop of blood on a slide with a needle in the manner recommended by Christophers and Stephens. It is not always easy, however, to regulate the width of the film. It is often too wide and its edges lie outside the area under the cover-glass. This is a disadvantage when leucocytes have to be counted, as they have a way of getting to the edges and ends of the film, and consequently, if the film should be too wide a large proportion of the leucocytes would not be under the cover-glass. This disadvantage can, to a certain extent, be overcome by practice, but not always. When the patient is very anæmic, and the blood thin and watery, it is often surprising to note how quickly a drop of blood runs along the needle to the very edges of the slide. In these cases the tissue paper method is the best, as the width of the film can be regulated by the width of the strip of paper.

As cover-glasses soon devitrify in this climate it is necessary to adopt some means of preserving them. I put them into a wide-mouthed bottle of ether as soon as received from the optician. This removes all grease and apparently preserves them as well as anything else and they are always ready for use.

SPIRRILLUM FEVER (RELAPSING OR FAMINE FEVER).

By CUTHBERT CHRISTY, M.B., C.M.Edin.

THE following notes on an epidemic of spirillum fever in India may be of interest.

In September, 1900, while I was stationed at Ahmednagar, perhaps the healthiest and nicest station in the Bombay Presidency, one of the circle inspectors reported through the Mamlatdar, Taluka Nugar, that an unusual amount of sickness was prevalent in the small village of Jeur, about ten miles to the north. This village, I was told, had suffered from plague during the previous year (1899). As Acting Civil Surgeon of the district I considered it my duty, therefore, to at once make a visit of inspection, the District Medical Officer being in another part of the country.

On arriving at the village, I found it to be of the typical Indian agricultural sort—a cluster of flat-roofed houses, built of sun-dried mud, many of them

only ventilated and lighted by a low, dog-kennel-like doorway. Its population was judged to be about a thousand.

I first proceeded to make an inspection of the sanitary condition of the place, and found it on the whole cleaner than many similar places I had visited. An Indian village of this type has one thing in common with Irish villages—the close association of the inhabitants with their domestic and other animals. In this case, instead of pigs, cows and buffaloes, old and young, blocked up the narrow passages, or were stabled almost in the living rooms. I continually had to climb over heaps of stable refuse before reaching the doorways.

A police officer, who had arrived at the village before me, had prepared a list of persons who were sick, so after my inspection of the surroundings I commenced to visit these one by one. In almost every instance I found persons suffering from "fever," or the results of it. After having examined a number of the cases it was evident that it was not malaria, but spirillum fever that I had to deal with. Jaundice was frequent, whilst injected and ecchymosed conjunctivæ were prominent features. Histories of pains in the joints, severe fever with more or less weekly intermissions, and the occurrence of many cases of parotitis, strengthened the diagnosis. Only in two or three cases, at the time undergoing a relapse, did I find an enlarged spleen; but slight enlargement of the liver, with tenderness over that region, was common. There were nowhere any signs of plague. The largest proportion of the cases seen were males, but probably the females had not been notified in the same proportion as the males.

In studying the list afterwards I concluded that the greater number of the other diseases met with in the course of my inspection were in all probability the results of spirillum fever, for example, "mumps," bronchitis and chest trouble generally, acute nephritis in three children, complaints of "rheumatism," &c. Only in one instance did I find what might have been a case of malaria.

Apparently distinct centres of infection were to be noticed, almost all the occupants of certain blocks of houses were suffering from the "fever" in one stage or another, or the results of it.

After my inspection, having done my utmost to ingratiate myself with these people, I tried to persuade some of the cases to allow me to take some blood smears. As I expected, all refused. A period of commotion and excitement began amongst the assembled multitude, and I soon saw that I had fallen in their estimation. Amongst superstitious people of this sort, particularly in some isolated rural districts, it is imperative that the official should raise no suspicion of inoculation or like procedure. Inoculation under these circumstances can only be carried out when the community is influenced by elders or natives sufficiently educated to appreciate its benefits, and who set the example by being inoculated themselves. Serious riots, in which murder has played a part, have resulted in India from attempts to enforce inoculation.

The taking of blood slides is naturally looked upon as some form of inoculation, and I have on several

occasions found myself in situations which threatened to become serious owing to my attempts to procure them. However, in this case I was determined to get my slides. After much talk and explanation, and having demonstrated my process upon my own finger, and upon the ear of a sepoy, they at last began to give in. I then made an offer of Rs. 2, and in the end I was allowed to operate upon a boy about 20 years of age, living in one of the infected blocks, whose temperature was 103°, and whose appearance was typical of most of the other cases. From him I procured two smears, and, as no one else was willing to offer himself, I made the best of my way back to Ahmednagar, glad to be out of the village, with its dirt and sickness, and away from the evil looks of its inhabitants.

On reaching my bungalow and staining the two slides with gentian-aniline-violet, I found them both full of the *spirillum Obermeieri*. This fact, taken in conjunction with the chief symptoms I have mentioned, is, I think, sufficient to establish the occurrence at Jeur of an isolated epidemic of relapsing fever. Whether it had been imported from Bombay, where the disease is endemic in certain quarters, I did not discover. Jeur is not situated in a district much influenced by famine, and its people, though exceedingly poor agriculturists, were not in great poverty, judging by their general appearance and that of their bazaar.

Close by the largest well, evidently the centre of washing operations, I found a grass-grown puddle containing an enormous collection of larvæ of some species (not identified) of *Anopheles*. I saw no signs of *Culex*. Whilst visiting the houses I was only able to find two or three *Anopheles*, and these I could not secure; yet there must have been numbers of them, although the inhabitants all seemed to agree that there were none.

There was no apparent relation between the infected blocks of houses and the situation of this breeding place of *Anopheles*. The bed bug was everywhere, as it usually is in such situations.

As it is possible that the bed bug may have some close connection with the transmission of spirillum fever, the following additional notes may be of interest here.

It is not uncommon during a morning's ramble through the narrow streets of Mandvi or Omerkadi, two of the most overcrowded quarters of Bombay city, and where spirillum fever seems to be endemic, to see a woman come into the middle of the roadway and tap a piece of wood upon the ground. On investigation it will be found that the piece of wood is a bug-trap. It is generally about a foot long by an inch or so square, and has several deep cuts made longitudinally on two opposite sides of it with a saw. Whether any special wood is used I do not remember. These bug-traps are placed by the better-class natives beneath their bed-clothes at night, and in the morning the bugs retire to the slits as being the most convenient and nearest shelter for the day. But a Hindu is debarred by his religion from killing any animal whatsoever. The good housewife, or one of them, therefore, instead of plunging the trap into hot water, takes it into the street and taps out the

bugs upon a stone, leaving them to crawl back to the nearest house. The act, like most acts connected with Hindu worship, savours greatly of a certain biblical character, who, wishing to call attention to his piety, prayed aloud at the street corner.

Whilst working in Bombay it struck me that these traps might be very conveniently used for collecting bugs for experimental purposes. I procured, therefore, a number from the bazaar, and had them placed in the beds of persons suffering from spirillum fever, and thus collected a quantity of bugs for examination. From one case, in whose blood I had the day previously found the spirillum, I obtained during the third night of the relapse some thirty bugs. These I kept, trap and all, in a Winchester quart bottle; and every day, with the exception of two, for a fortnight, I fed one of the bugs upon my arm, at the same time examining one which I had not fed. The result, however, was negative as far as my health was concerned.

By reason of its strong chitinous exterior the bug, like the flea, is extremely difficult to dissect and examine, and any suggestions tending to simplify the proceeding will, I feel sure, be welcomed by many besides myself.

MALARIAL FEVER AS MET WITH IN THE GREAT LAKE REGION OF CENTRAL AFRICA.

By ALBERT RUSKIN COOK, M.D., B.Sc.Lond., B.A.Camb.
Late Scholar of Trinity College, Cambridge.

PROLEGOMENON.

THE importance of malarial fever in this country to every immigrant or native can scarcely be gainsaid. Regarded from a medical, social, utilitarian, or even political, point of view, it bulks large in practical every-day life. The country may be on the one hand, suddenly deprived of the services of a sagacious statesman at a political crisis, or on the other hand, its population may be steadily sapped by the ravages of this minute but almost invincible foe. The high death-rate of malaria amongst Europeans is well known. Practically no one who resides in the country for any length of time altogether escapes it, and even amongst the natives, as will be subsequently shown, it claims a very large number of victims, whilst the morbidity caused by it is even greater. Under these circumstances then, every item of intelligence that can be turned to useful account in attacking this scourge of man should be gathered up and placed on record. If tuberculosis has been proved to be a preventable disease, malaria must fall into the same category, and whatever method or combination of methods may be employed the struggle should never be regarded as hopeless or relinquished as Utopian.

It would be quite impossible to describe adequately, in such a short compass as the limits of this paper impose, the whole subject of malaria as met with in Central Africa. To do so would fill a volume several hundred pages long. All that the writer aims at is recording such points as may throw light on the more serious attacks of fever. Nearly five years' continuous

residence in tropical Africa, though too short for a comprehensive study of the disease, has at least afforded scope for recognising some few main principles. In the practice of the hospital to which I have the honour of being attached we see about 1,200 cases of fever yearly, and this affords an ample field of research. It is only fair to state that the commencement of the work was beset with difficulties. When I left the coast in 1896 it was a three months' march into the interior to reach Uganda, and delicate instruments are not improved by being carried for such long distances on porters' heads. Fortunately the railway, now approaching completion, will alter all that. The publication of such a paper as the *JOURNAL OF TROPICAL MEDICINE*, the foundation of two schools of Tropical Medicine in England, and the advent of many excellent books on tropical diseases, all testify to the increasing intelligence with which the English people are endeavouring to grapple with the great malaria problem.

GEOGRAPHICAL EXTENT OF RESEARCH.

The following observations were made in a region bounded roughly by the Victoria Nyanza on the south, the Nile on the east and north, and the Albert Nyanza, Ruwenzori Mountains (Mountains of the Moon), and Albert Edward Nyanza on the west. The immense majority of cases of fever occurred amongst the Baganda, the hospital of which the writer is in charge being situated at Mengo, the capital of Uganda; but considerable numbers of Banyoro, Batoro, Banyankole, and Basoga were seen in journeys through the respective countries of Unyoro, Toro, Ankole, and Usoga. The number of cases of fever observed in Europeans was of course much less, the European population of Uganda being confined to missionaries, Government officials, and traders.

NATURE OF COUNTRY.

Uganda is a country lying like a cap immediately to the north and north-west of the immense body of fresh water known as the Victoria Nyanza, which is situated over 3,800 feet above sea level, and has an area equal to twice that of Belgium. The Equator crosses the lake near its north shore, and thus runs through the south-west part of Uganda. The latter is very hilly, the hills being largely of the older geological formations, and rising from 100 to 400 feet above the level of the surrounding country. Separating each hill from its neighbour is a sluggish stream or swamp, choked with a dense growth of papyrus, so that a road from one village to another is a monotonous succession of hill and swamp alternately, the road being generally taken over the highest part of each hill. The swamps are crossed by rough causeways. The heat is not at all excessive, the thermometer in the shade of our heavily-thatched houses seldom rising above 86° F., even at mid-day, and what is more important remaining wonderfully uniform, only occasionally registering a temperature of under 76° F.

The year is divided into two rainy seasons, alternating with two dry ones; the former commencing in March and September respectively, and lasting some three months, but rain falls in every month all through the year.

The red deeply ferruginous soil is very productive, and but little labour is needed to raise food. The staple food of the country is bananas. With the exception of the chiefs and more wealthy men, who often get meat and milk, the immense bulk of the population live on nothing but steamed and mashed plantains, which they call "emere," and on sweet potatoes. Indian corn is regarded as a delicacy. Much "mwenge," as the native beer is called, is manufactured by fermenting banana juice and is freely drunk. All adults are well clothed with native manufactured bark cloth or imported American sheeting or linen. Children to the age of four or five are allowed to run about naked, and often get chills in consequence. They are frequently suckled for eighteen months or two years. The huts are not built close together, but scattered throughout their gardens. They have no windows, being of the usual conical shape, and ventilation is very defective. In many places the level of the subsoil water is only just below the surface; much of the soil is clayey. The prevailing wind during the daytime is from S. to N., during the night from N. to S.; this is doubtless owing to the proximity of the lake.

MORPHOLOGY OF THE MALARIA PARASITE.

Judging from the published descriptions of the malarial parasite in Davidson, Manson, Coles, &c., there is at least one species met with in Central Africa that is not described as occurring in Italy, Algiers, or China. Nor is this to be greatly wondered at. Africa is a large place, and if we except the admirable investigations carried out by Drs. Daniels, Christopher, &c., round Lake Nyassa and at Sierra Leone, but little has been done to elucidate the varieties of fever existing in Central Africa. It is obvious that the truth will only be arrived at by many workers in different places, each stating exactly what they find. When I left England in 1896, the only book available to me on the subject was Davidson's well-known work on Tropical Diseases. Since then books on malaria have greatly multiplied, and thanks to such books as Manson's, every practitioner in the tropics has, if he cares to take a little trouble, an excellent opportunity of enriching his own mind and adding to the general store of knowledge. Let me then first briefly indicate the agreements between such observations as I have been able to make, and the, what I may call, classical accounts of the parasites met with in the text-books, proceeding subsequently to point out the differences.

METHOD OF PREPARATION.

(1) *Fresh Specimen.*—The finger-tip, after being cleansed, is pricked with a sharp sterilised needle (preferably a surgeon's Hagedorn needle), a minute drop of blood exudes, which is wiped off, and the next which wells out is touched with the centre of a clean cover-slip in such a way that it does not come in contact with the skin, and the slip is then lowered gently on to a perfectly clean slide. The preparation, either at once, or after being ringed with vaseline, is examined first under a low power to choose a part where the red blood corpuscles are disposed singly and not in rouleaux, and then a high power is turned

on to study more minutely the structure of any plasmodia present. For the latter purpose I generally use a $\frac{1}{2}$ in. oil immersion and No. 3 eye-piece (Swift). This gives a very clear magnification of 730 diameters. A No. 5 eye-piece and lengthened tube increases this to 1,627 diameters if necessary.

The most important practical point in the above is to get really clean cover-slips and slides. This seemingly simple process is often most difficult. Boiling the cover-slips in 10 per cent. sodium bicarbonate solution, then rinsing in several changes of water, gently warming in strong sulphuric acid, and again thoroughly rinsing and finally keeping in absolute alcohol is perhaps the best of many methods. For use the cover-slip may be held for a moment in the flame of a spirit lamp to burn off the alcohol. Slides may be cleaned in the same way, but do not need to be kept in alcohol.

(2) *Stained Specimens*.—The drop of blood that wells out from the pricked finger is touched with a fine piece of cigarette paper about $\frac{1}{4}$ in. from its end. The latter is then touched to the surface of a clean glass slide, the droplet of blood allowed to spread out, and the paper drawn by its uncharged end along the surface of the slide. A little practice serves to produce a blood film in which the corpuscles are disposed in a perfectly even and regular manner. The film is then fixed by the slide being immersed for five or ten minutes in absolute alcohol contained in a wide-mouthed bottle. The slide is removed, allowed to dry, and a few drops of a stain consisting of borax 5 per cent., methylene blue 2 per cent., in aqueous solution blown gently on from a pipette, and allowed to remain on for about thirty seconds (Manson's method). The excess of stain is then washed thoroughly off with water, the film allowed to dry, a drop of xylol-balsam dropped on, and a cover-slip applied. If the specimen be required merely for diagnostic purposes I generally mount in cedar-wood oil. Stained specimens have considerable advantages over fresh. You need carry with you only slides, cover-slips, cigarette papers, and needles—no small advantage when suddenly summoned fifty miles in this country, where everything has to be carried on men's heads. The films once made keep indefinitely, while the fresh specimens must be examined within a short time after their preparation. The stained films show the minute structure of the parasites, and what is more important, by this method one can survey, if the film be a good one, an enormous number of corpuscles in a very short time; the stained parasites standing out very clearly. At the same time fresh specimens alone show the amoeboid movements of the intra-corpuscular form of the parasites and any phagocytosis on the part of the white blood corpuscles that may be going on, or the actively motile flagellated bodies.

The varieties of malarial parasites are usually divided into:—

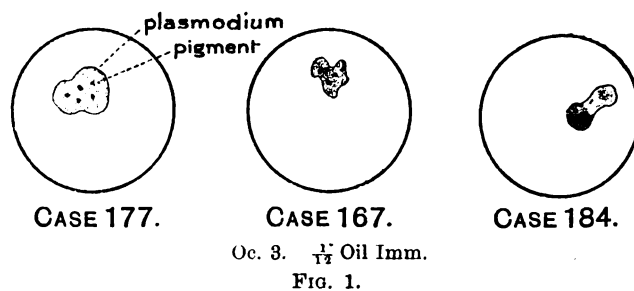
| | | |
|---------------------|---|------------------------|
| CLASS I.—Benign | { Quartan Tertian } | Do not form crescents. |
| CLASS II.—Malignant | { Quotidian—pigmented Quotidian—unpigmented Tertian } | Form crescents. |

I may briefly dismiss the first class by saying they

are met with, though not nearly so commonly as the so-called malignant forms. A double tertian benign infection gives rise to the ordinary quotidian ague. A single tertian intermittent is the commonest type of fever amongst Europeans here. Quartan infection is distinctly rare. Details will be given under the clinical types of the disease.

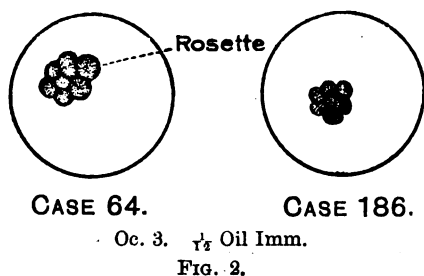
As regards the members of Class II., we notice one marked difference. Crescents are very rarely seen, though the flagellated forms are not uncommon. The obvious deduction seems to be that the latter are derived from some other form of the parasite than the crescent. As the parasites belonging to the first group differ in no respect from those found in more temperate climes, I will not describe them.

In a typical attack of severe African fever, characterised clinically by only imperfect remissions of temperature, an anxious "facies," incomplete rigors, or indeed mere sensations of chill, a non-perspiring skin, and often marked mental oppression, severe headache, and pains in the back and limbs, films prepared from the blood at any time of the day will, on being suitably stained, show the following appearances: Parasites will be noticed within a varying proportion of the red corpuscles of the blood. From 1 to 5 per cent. of the latter may be invaded, or even as high a proportion as 10 per cent. The proportion of attacked corpuscles in the peripheral blood seems, however, to be no index of the severity of the disease. In one of my cases, a European, a comparatively mild attack, though characterised by severe headache and subsequent feebleness, showed an infection of about 10 per cent. of the red blood corpuscles, while a fatal case examined only a few hours before death showed very few intra-corpuscular parasites. As regards the result of the administration of quinine, it is often possible for days, in some of these cases, to discover many intra-corpuscular parasites, even while the patient is soundly cinchonised, and this doubtless explains the well-known clinical fact that even under the full doses of quinine the fever yields very slowly, the temperature reaching the normal line, it may be, only after eight or ten days' treatment.



Comparing the results of many observations I may say that the earliest stage is that of a minute unpigmented intra-corpuscular body. In a fresh specimen of blood (I cannot do better than quote Manson's description of it) it looks "like a little washed out smudge of a dirty white paint." This slowly increases in size, but never (in the form of fever I am now describing) attains a very large size, rarely exceeding in diameter one-fourth to one-third the diameter of a

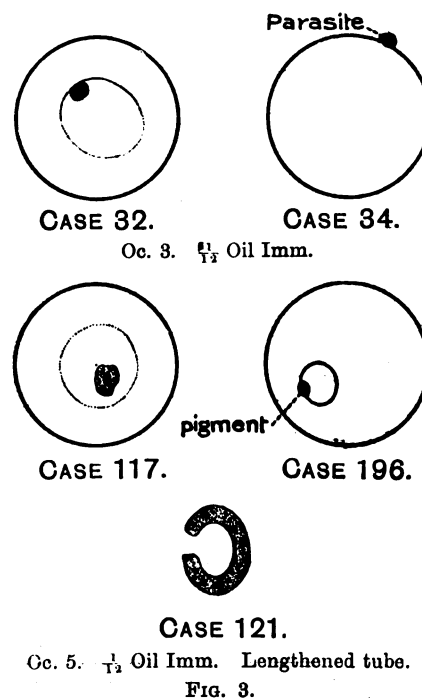
red blood corpuscle. As it grows it becomes pigmented, the pigment being, in methylene blue preparations under a high power, of a reddish-brown colour and not intensely black like ordinary melanin. The pigment is in the form of fine dots or granules but is never very abundant. When full grown the parasite proceeds to sporulate; this process, however, takes place but sparsely in the peripheral blood, but time and patience will enable one to see very typical examples. The mature sporocyte consists of a varying number, 12 to 20 spores, arranged in a very definite mulberry-like mass, reminding one very much of the morula stage of the segmenting ovum. In the cases I have observed I should like to emphasise that the mature sporocyte by no means fills the whole red blood corpuscle. Usually the spores, which stain intensely, are too heaped up to see the pigment in the centre. The corpuscle breaks up and the spores set free, that is, such as escape being engulfed by the watchful phagocytes, proceed to attack fresh blood corpuscles.



Another feature of this irregular remittent type of fever is that parasites of nearly every age are met with in the same specimen, or perhaps two well-marked groups may be seen in varying proportions, *e.g.*, a large majority young, but a substantial proportion full grown.

Careful focussing shows that the shape of the young intra-corpuscular parasite is most variable, possibly due to its being fixed when in active amœboid movement. A very common appearance is to see a small sphere as though it were gummed on to the periphery of a red blood corpuscle, or lying just within the circumference. In its youngest form the whole parasite stains intensely, later on a flange or projection of lightly-stained protoplasm may be seen at one side, or a comparatively unstained body may surround the central darkly-stained part. Later again granules of pigment may be seen scattered over the whole stained surface. In a good many cases, too many to be merely accidental, I have noted blocks of reddish pigment lying eccentrically in the red blood corpuscles. It is just possible that these may have been deposited on the corpuscles and are not manufactured out of its substance. Some varieties seem to be quite unpigmented in the peripheral circulation. Probably the sporocyte matures and segments in the spleen, bone marrow, &c. The small size and scanty pigment in the earlier stages of the plasmodium render it difficult to see them in fresh preparations, when the parasites are few in number. For this purpose stained films have a distinct advantage. Thus in one case (No. 117 in my Series) a very perfect fresh specimen showed no parasites, though hundreds of cor-

puscles were carefully scrutinised under a high power. There had been no rigor and it was the first twenty-four hours of the fever (T. 100). A stained film taken at the same time showed large numbers of exceedingly small unpigmented parasites. Another common form, and one that quickly disappears under the influence of quinine, is the so-called "signet ring" parasite. In this kind the parasite assumes a delicate ring-like shape, with an eccentrically placed bulge which stains deeper than the rest. It may be unpigmented, but is usually studded with pigment granules. In a few instances I have found the same body apparently free in the plasma. Sometimes a horse-shoe shape is assumed.



MULTIPLE INFECTION.

Not unfrequently two or even three parasites may be seen in one corpuscle; this indicates a grave attack. As a broad rule, if the blood be examined when the temperature is high only the smallest kind of parasite will be seen.

(To be continued.)

DR. S. CARSON RYNHARDT in his book, "With the Thibetans in Tent and Temple," mentions that there is no legitimate medical science in Thibet.

For headache large sticky plasters are applied; to relieve rheumatism a needle is plunged into the patient's arm or elbow. Teeth are extracted by means of a string, with the result that very often a part of the jaw bone is dragged away simultaneously. Persons suffering from the stomach are energetically belaboured, or a piece of wick steeped in boiling butter is applied to the part.

The remedies to be taken internally are often composed of a pellet of paper on which a prayer is inscribed. If this remedy takes no effect a second pellet made of the bones of a pious priest is administered.

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THE

Journal of Tropical Medicine

FEBRUARY 1, 1902.

LESSONS TO BE LEARNT FROM PROFESSOR BRUNO GALLI-VALERIO'S PAPER.

THE article in this issue of the JOURNAL, by Dr. Bruno Galli-Valerio, should be studied carefully as an object lesson in scientific investigation. Dr. Galli-Valerio has not succeeded in showing, in fact he makes no pretension to do so, how plague is transmitted to man, nor even that the rat plays an important, or any part, in the conveyance of plague from man to animals. His article, however, is a clear exposition of our knowledge of the relations of rats to man as regards plague infection, and he demonstrates conclusively at what a fine point of scientific precision the investigation has arrived.

After discussing several plausible theories, showing where they are weak and where they are scientifically valueless, he brings us by his pro-

cesses of reasoning to this point, namely, that to prove the theory of the power of infected rat-fleas to transmit the disease to man, man must suffer himself to be bitten by infected fleas. This of course is, has been, and will remain the halting point in many investigations of the kind; and, as in several recorded instances we find men willing to submit themselves to be experimented upon, so in this instance, Dr. Galli-Valerio is willing to become the subject of experiment, if such is considered necessary.

But important although these points may be, it is not on that account we wish to direct attention to this article. It is because Professor Galli-Valerio has given us information in regard to plague in a direction other than clinical and bacteriological. These have been the aspects of plague which have been dealt with by writers and observers well nigh exclusively, ever since 1894, when the modern pandemic declared itself in Hong Kong.

It is now eight years since Aoyama, Cantlie, Kitasato, Lowson and Yersin, gave us definite information concerning the signs and symptoms, the anatomical characters, the bacteriology, and even the serum-therapy of bubonic plague, and beyond the discovery of pneumonic plague by Childe in 1896, and important work by Haffkine and Hankin in India, subsequent investigators have practically been treading the same ground. They have given us stacks of literature—clinical, pathological, and statistical—but the additions to our knowledge of plague have been mostly details.

The various foreign scientific expeditions to India were disappointing, they but reiterated what was already definitely known, and for the most part wasted both time and money. Reports on plague may be necessary for Governments, but the men employed in drawing them up have often to spend their time and intellect on scientifically useless work. Valuable men, capable of a better and higher order of work, are chained to the slavery of routine to no purpose, and are compelled to do work which their training and capabilities pronounce to be unworthy of their attention.

Dr. Galli-Valerio's paper, as Dr. Manson says in his comments on the paper, "shows on what a slender basis of fact a catching theory may be founded and how it may gain popularity and even acceptance." According to Manson, the paper also indicates "the disinclination for workers in plague to step outside the well-worn ruts of the clinical observations and the bacteriology of the disease; for, except for two or three inconclusive experiments, nothing has been done to exploit the rat-flea theory."

The cause, the origin, or the mode of diffusion of disease, after the clinical facts and the bacteriology of the disease are known, should be the work which scientific investigators should take up, not as a side issue, but as the kernel of the question. The prophylaxis naturally follows upon the discovery of the cause or of the means of transmission. Dr. Galli-Valerio's paper will serve as a guide in future to medical practitioners who wish to investigate the causes of disease, and there is no medical man, however isolated, who cannot devote his attention to the elucidation of the cause of disease on the lines laid down by the writer of the article in question.

Article for Discussion.

ON THE DANGER OF SUBCUTANEOUS INJECTION OF QUININE.

By J. PRESTON MAXWELL, M.B., F.R.C.S.

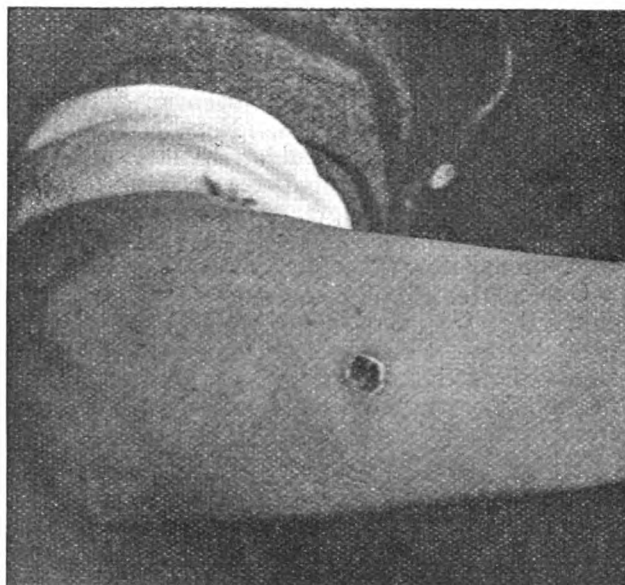
DR. TOWNSEND'S letter and Mr. Cantlie's comment on the same *re* the subject in hand, raise a matter which is of the utmost importance to those working in malarious countries. And as the writer has had personal as well as practical experience of the same, a few remarks on the subject may not be deemed out of place.

Some seven years ago, when preparing to come abroad, my father, Dr. Maxwell (late of Formosa, Japan), and I carried out some experiments on this matter, using most of the available salts of quinine, including an oleate and a borate, with the intention of determining the safest and most pleasant way of administering this injection. On one occasion my father produced the gangrenous patch on his forearm which is shown in the accompanying photograph, and on another occasion I spent a week in the isolation ward of the

hospital, where I was at that time studying, with a curious form of cellulitis of the arm.

As I believe both of these accidents to have been due to the use of a *too concentrated* solution, I will briefly give the details of the last named. On one evening, about 5 p.m., I injected into the extensor surface of my left forearm a solution of 5 grs. of the hydrochlorate dissolved in the absolute minimum of water. The solution was injected warm.

After half-an-hour I had slight cinchonism and my arm began to swell, and by next morning was swollen up to the shoulder and very painful. There was no tenderness in the axillary glands and the temperature was 99° F.



Although the fullest antiseptic precautions had been taken I feared that I must have poisoned myself, and went off to hospital. My temperature never reached 100° during the whole illness, and in a week my arm was of its normal size.

A small cold abscess formed at the point of inoculation, was opened, and a few minims of pus let out. There has been no further trouble from the same. Agar-agar and gelatin tubes were inoculated from this pus but failed to yield any growth. My own opinion was, and is, that the abscess was due to chemical, and not microbic action.

My father, who used much the same injection at the same time, had a similar train of symptoms, but these were not so severe, and the injection did not produce any tissue death. Since that time I have always handled hypodermic injections of quinine with respect, but am by no means willing to abandon the method, as its efficacy is undoubted, and where quinine is not tolerated by mouth or rectum it may prove the

only possible method of introduction. For the last three years I have given quinine hypodermically in the following way :—

(1) The injection should be intramuscular, not subcutaneous.

(2) The gluteal, scapular and deltoid are the muscles best suited for this method, and especially the first named.

(3) I use an injection consisting of 6 grs. of the hydrochlorate (Burroughs, Wellcome and Co.'s tabloids) to forty minims of boiled water. This solution is brought to boiling point and allowed to cool. The essential point is a large dilution of the acid salt.

(4) After injection, which is always painful, I paint Linimentum Iodi over the point of injection and the surrounding area. This plan was recommended very warmly by Dr. Sims, of the Congo, who was a strong advocate of the method.

As to results, I have never seen any untoward occurrence by this method. None of the injections gave rise to any trouble whatever, although in one case, a debilitated youth with malarial ascites, I injected 6 grs. every two days for a fortnight into various muscles, taking no situation oftener than once in six days.

One matter should be borne in mind. I do not think that injections are absolutely safe in the case of those suffering from any form of acute septic ulceration, as in these cases there is a possibility of the area of injection being infected from within.

With reference to the question of sepsis. The more irritating an injection, the more damage does it do to the tissues and the more careful must one be with one's antiseptic precautions.

I should not wish to charge all those who are unfortunate enough to meet with suppuration in the use of this method with being careless or dirty, but I do emphatically protest against the argument from strychnine injections. In many cases the wonder is not that these injections do not cause suppuration, but rather that they do not more often do so.

And I have very little doubt that if I were to inject quinine as I have more than once seen strychnine given I should have no lack of suppuration. I quite agree with both Dr. Townsend and Mr. Cantlie that when suppuration does occur from a quinine injection it is apt to be tedious and difficult to cure.

NOTE BY MR. CANTLIE.

If the abscesses which follow quinine and strychnine subcutaneous injections are to be attributed largely to sepsis, how is it that abscesses or cellulitis are so seldom seen after hypodermic injections of morphia, or morphia and atropine? The number of hypodermic administrations of morphia compared with those of quinine, strychnine, &c., may be safely said

to be 1,000 to 1, yet it is quite the exception for morphia injections to be followed by any untoward sequelæ. Is it that extraordinary precautions are taken against sepsis when morphia is used? I think not; every practitioner is well aware that, even before Listerism taught us to be more careful in even our minor manipulations, hypodermic injections of morphia were abundantly practised without necessarily local subsequent inflammations following. Nor is it to be attributed to carelessness on the part of the practitioner in the tropics. Every one who *knows* the medical practitioner in the tropics is well aware that he is certainly not behind his fellow practitioners at home, and were I to push comparisons between the two my testimony would be certainly even more pronounced than the negative statement that "he is certainly not behind." I agree with Dr. Maxwell that the abscesses, &c., which follow quinine injections may be attributable to "chemical, and not microbic action."

News and Notes.

MESSRS. J. DEFRIES AND SONS, Limited, 147, Houndsditch, London, E.C., the well-known manufacturers of the Pasteur Chamberland Filter, of the Equifex Disinfectors, steam stoves, and of various other appliances, which are daily proving their usefulness in public health matters, are distinguishing themselves in quite another direction. It appears that this enterprising firm produced some of the most important public decorations and illuminations during the reign of Queen Victoria, and in fact as long ago as the coronation of Her Majesty contributed largely to the decorations of the streets of the metropolis on that important public occasion.

That their abilities as public decorators and illuminators have not been forgotten are amply testified by the fact that Messrs. Defries have been appointed decorative illuminators to His Majesty, King Edward VII.

That the work of the firm in this direction is not detracting from their efforts on behalf of the public health, is vouched for by the continued confidence their appliances command. In testimony thereof it may be remembered that the *Ophir*, with the Heir Apparent to the Throne on board, was supplied with their Pasteur filters.

In tropical countries, the practitioner is often at a loss for consulting-room appliances in the way of light suitably and conveniently arranged for ophthalmic, aural and laryngeal examinations, owing to the want of electric light or gas in his vicinity. Messrs. Defries, by their attention to the manufacture of illuminators, are likely to be called upon by medical practitioners in warm climates to supply their wants in this direction, and we have no hesitation in saying that their requirements will be fully and satisfactorily met.

Current Literature.

Papers in the British Medical Journal of January 25, on Tropical Diseases.

I.—THE OPERATIVE TREATMENT OF LYMPHANGIECTASIS OF FILARIAL ORIGIN.

Lt.-Col. J. Maitland, I.M.S., Professor of Surgery, Madras Medical College, draws attention to the marked relief he was able to give in several cases of lymphangiectasis of the glands of the groin by removal of the mass. A number of similar operations have from time to time been reported, more especially by surgeons in Madras, and the benefit to the patients has, in practically all cases, been considerable. In some there has been entire cessation of attacks of fever and pain, and in several the disease would seem to be eradicated.

The success of these operations forms a welcome addition to the treatment of filarial infection and its sequelæ; and although the *rationale* of the operation is at present somewhat obscure, practice has proved that it is often successful in relieving the patient from the periodic attacks of fever and pain. In none of the cases recorded has lymphorrhagia, or the formation of lymphatic fistulæ followed the operation; nor has there been any evidence of a tendency to septic inflammation after the operation.

Lt.-Col. Maitland states, in his description of the operation, that he removed the entire mass of groin glands; where this is possible it is conceivable that his contention, that "the first principle of preventive treatment in such cases should be to endeavour, if possible, to remove the parasites," places the operation on a sound basis. It is seldom, however, the groin glands are alone enlarged, and where the enlargement extends along the iliac vessels the good to be gained by removing the groin glands only, is more difficult to understand. Perhaps Lt.-Col. Maitland's remark that "it is extremely probable that the stasis of lymph which follows ligation of the vessels results in the death of the parasites," even when centripetally placed, is correct. It is, at any rate, a distinct advance towards affording a scientific explanation of the good resulting from an operation which has heretofore been condemned on account of its empiricism.

II.—OBSERVATIONS ON HUMAN FILARIASIS IN TRINIDAD, W.I.

George A. Vincent, M.B. C.M., Trinidad Medical Service, gives the following table showing prevalence

| Race | Number Examined | Number with Filaria | Percentage with Filaria | Number with Elephantoid Disease | Percentage with Elephantoid Disease |
|------------------|-----------------|---------------------|-------------------------|---------------------------------|-------------------------------------|
| Blacks (negroes) | 323 | 16 | 4.9 | 21 | 6.5 |
| Whites .. | 55 | 6 | 10.9 | 8 | 14.5 |
| East Indians | 122 | 8 | 2.4 | 4 | 3.2 |
| — | 500 | 25 or 5 % | — | 33 or 6.6 % | — |

of filaria and filarial disease in 500 cases taken indiscriminately from persons dwelling in Trinidad.

The majority of the whites examined were natives of Barbadoes and Demarara, where filarial ailments are frequent amongst the white population.

In his remarks upon the prophylaxis of filarial ailments Dr. Vincent states "that filariasis is transmitted through mosquito bites, admits of little doubt, whatever may be the other means by which infection may be carried, and protection from infection practically means protection from mosquitoes." It may be possible to rid a locality of *Anopheles*, as they require special conditions for breeding, but as culices breed in any collection of water, their extermination is no easy matter. "Much may be done even in the case of *Culex* by protecting our stored water and frequently emptying all collections of water, so as not to allow sufficient time for the larvæ to mature. Meanwhile the only safeguard is to be found in the habitual use of mosquito nets, especially when in the neighbourhood of known filariated individuals. If the malarial subject is a source of danger to those around him, a filariated individual is doubly so, for in his case the carriers of infection are drawn from the ranks of a very common species of mosquito, as well as from the rarer species to which we owe malaria."

PLAGUE.

PREVALENCE OF THE DISEASE.

INDIA.—During the weeks ending December 21st, December 28th, and January 4th, the number of deaths from plague throughout all India amounted respectively to 9,198, 8,005, and 9,556.

In the city of Bombay the deaths from plague during the three weeks in question numbered 175, 180 and 213 respectively.

In Calcutta during the four weeks ending January 4th there were 149 plague seizures and 136 deaths from the disease.

EGYPT.—During the two weeks ending January 20th 31 fresh cases of plague were recorded in Egypt and 25 deaths from the disease. The majority of cases occurred in the town of Tintah.

CAPE OF GOOD HOPE.—At Port Elizabeth and Mossel Bay 1 and 2 fresh cases of plague respectively were reported during the week ending January 11th. No deaths from plague were notified anywhere in Cape Colony.

MAURITIUS.—During the weeks ending January 16th and 23rd the number of fresh cases of plague amounted to 27 and 14, and 21 and 7 deaths from the disease.

UNITED STATES OF AMERICA.—Up to November 4th, 1901, as many as 51 cases of plague were reported in San Francisco.

At Utah, Salt Lake city, on November 2nd, a death from plague was reported.

CANCER AND MALARIA.—Professor Kruse, from a statistical study, reaches the conclusion that, in climates where malaria is prevalent, cancer is not often found. He does not attribute this to the presence of malaria, but rather to an immunity of the tropical races to cancer.—*Münchener medizinische Wochenschrift*, November, 26th, 1901.

NO YELLOW FEVER IN HAVANA.—Major W. C. Gorgas, Chief Sanitary Officer at Havana, in his report to Governor-General Wood for November, says: "During the month we have had no cases and no deaths from yellow fever. This can be said of no preceding November since 1762. Last year we had, during this month, 214 cases and 54 deaths. This year the last case of yellow fever occurred on September 28th; that is, we have gone over two months without a single case or death belonging to Havana. I consider this a demonstration that Havana has at last been freed from the infection of yellow fever. It must be remembered that October and November are the months when yellow fever is rife in Havana, and that, for the past century, there has never been a day during the two months when there were not many cases in the city. This result I consider due to the system introduced last February of killing infected mosquitoes in the neighbourhood of each point of infection as it developed."

A CONGRESS ON PELLAGRA.—A national Italian Congress on Pellagra is announced for May, 1902. Papers will be presented regarding etiology, treatment, and means of prevention.

BABOOL LEAVES AS A CURE FOR HYDROPHOBIA.—The terrors of hydrophobia and difficulty of a cure—the Buisson bath system not being resorted to as widely as its merits deserve—are sufficient reason for making known any remedy that seems reasonably well attested. Dr. J. E. Chamarette (retired surgeon, H.H. the Nizam's artillery, regular troops), residing at Hyderabad, Deccan, has made the following communication to *The Indian Medical Gazette*, November 6th, 1901: "It is now over twenty years that I have been administering in all cases of dog bite, be it inflicted by seemingly good healthy animals, or dogs with unmistakable signs of rabies on them, the juice of the babool leaves. This plant is widely known and grows commonly about gardens and all around the jungles. (Native names of the plant—*Devanha babool*, Hindi; *Moorkie thoomha*, Telugu; *Pea vallume*, Tamil. Species of *Babulæ acacia*, *ferinacia*) I am extremely obliged and indebted to my much esteemed friend, Mr. Charles Prayero, of Hugheston, Hyderabad, a retired Extra Assistant Commissioner of the British Service, for placing me in possession of this invaluable drug and prophylactic, while he himself has with success followed this plan of treatment, I believe, for over fifty years. Previous to administering the Babool juice, I have the wound or wounds squeezed out of as much blood as possible, and thoroughly syringed and bathed with hot water, cleaned and dried; after which, with a nicely pointed piece of lunar caustic (*Argenti nitras*), every part and recess of the wounds are freely touched, probed and rubbed into satisfactorily. Dose of the Babool juice: the leaves should be washed in water and pounded in a clean mortar and strained through fine muslin. For adults, 1 or 1½ ounce of the juice given morning and evening on an empty stomach (no food, &c., to be taken until an hour after); juice to be administered

regularly for three days and prepared fresh on each occasion. Should nausea or vomiting occur, a table-spoonful of sweet tyre (milk curds) native name 'chuckka dhye,' should be taken to allay these symptoms. Dose for children of riper years, from ½ to ¼ the above quantity of juice. Diet to be observed three days only: boiled rice or wheaten cakes cooked without any salt, and eaten with milk curds, chuckka dhye, a little weak tea. Stimulants of any and every description to be strenuously eschewed."

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Archives Russes de Pathologie, de Médec. Clinique et de Bacteriologie.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Brooklyn Medical Journal.
Caducée.
Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Esercito.
Hong Kong Telegraph.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
Journal of the American Medical Association.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Medical Record.
Medical Review.
Merck's Archives.
New York Medical Journal.
New York Post-Graduate.
Pacific Medical Journal.
Polyclinic.
Public Health.
Revista de Medicina Tropical.
Revista Medica de S. Paulo.
Sei-i-Kwai Medical Journal.
The Hospital.
The Northumberland and Durham Medical Journal.
Treatment.

Notices to Correspondents.

- 1.—Manuscripts sent in cannot be returned.
- 2.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.
- 3.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.
- 4.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.
- 5.—Correspondents should look for replies under the heading "Answers to Correspondents."

The Journal of Tropical Medicine.

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In order to meet the constant enquiry for addresses of respectable firms catering for the various requirements so difficult to obtain abroad, we give a list of names and addresses which we trust will be found useful to our numerous correspondents and subscribers.

Original Communications.

SLEEPING SICKNESS IN UGANDA.

DR. PATRICK MANSON, C.M.G., F.R.S., in forwarding the accompanying letter and notes on "Sleeping Sickness in Uganda," observes:—

Some time ago you published a paper from Dr. T. Howard Cook, of Uganda, on the subject of sleeping sickness. I forward you a letter lately received from Dr. Cook, which bears out in a remarkable manner his gloomy forecast as to the probability of the extension of disease in that country. I duly received the slides of blood to which Dr. Cook refers in his letter. The films were rather defective as regards quantity of blood, a circumstance which to my mind quite explains the absence of filariæ in a proportion of the cases in which the parasite was found by Dr. Cook. Of the 26 slides I found *Filaria perstans* in the following: 3, 4, 12, 18, 14, 5, 16, 25, 19, 21, 9, 13, 15, and 17.

I notice in a recent publication by Portuguese investigators that they failed to find *Filaria perstans* in the considerable number of cases which they studied in Angola. Seeing that the parasite is very common in the neighbouring Congo district, and that I have found the parasite in blood from patients from San Salvador in Portuguese territory, and that it occurs in the Island of Fernando Po, I suspect that the failure of the Portuguese investigators was owing, not to absence of parasites, but to unsuitable technique.

I hope to send you for publication presently a translation of the Portuguese paper.

C.M.S. HOSPITAL, NAMIREMBE,
MENGU, UGANDA PROTECTORATE, E. AFRICA,
December 13th, 1901.

DEAR DR. MANSON,—I am sending you by this mail twenty-six smears taken from a series of con-

secutive cases of sleeping sickness that came to our dispensary for treatment. I no longer admit them to our hospital. All were from patients clinically perfect types of sleeping sickness, and in all except No. 11 (who died in hospital) I easily found the *Filaria perstans* after examining a film or two of the blood. In No. 11, though I frequently examined the blood, I did not find any worm. I shall therefore be much indebted, or a little surprised, if you find *Filaria perstans* in film No. 11.

The films are numbered to correspond with enclosed notes.

I hope soon to send you fifty films taken at random from apparently healthy natives, to see if *Filaria perstans* is found in the healthy natives in these parts.

The prophecy I gave at the close of my paper (published by your kind recommendation in the JOURNAL OF TROPICAL MEDICINE for July 15th), to the effect that we should soon have more abundant material to study, has unfortunately come only too true. On one island alone (Burrunga on V. Nyanza) over 200 natives have died of sleeping sickness, and in many places it exists in epidemic form. Then in the districts of Kyagwe, in Busoya, and latterly round Mengu, it is slaying literally hundreds. I see personally from four to six new cases every week at our Mengu Dispensary.

I shall be much obliged if you will kindly acknowledge the safe arrival of the films.

Anything in this letter that you think ought to go to the JOURNAL OF TROPICAL MEDICINE, I shall be much obliged if you will forward to the Editor under "Correspondence."

Believe me, yours sincerely,
J. HOWARD COOK.

NOTES.

F. P. = *Filaria perstans*.

(1) Levi Myukakitunzi: *F. P.* found. Came back from war in Nandi and got ill; pain in head and

chest; tongue tremulous; no salivation; no tremors of limbs; no glands in neck.

(2) Lukiya: Hospital cook; drinks Namirembe water; has phthisis; blood taken September 20th, 1901; illness began in August, 1901; thinks she is better; had great itching; better now; glands in neck.

(3) Zakayo: Child; very tremulous; tongue and body tremulous; F. P. found; glands in neck not enlarged.

(4) Tito: Illness two and a half months; tremors of tongue and limbs; F. P. found.

(5) Faïda: Lives at the "lubiri," Mengo; ill two months; F. P. found; glands in neck not enlarged; obviously drowsy.

(6) Yokana: Pain in chest; came back from Nandi war ill with disease; not drowsy; says he does not sleep in daytime; skin irritable; rubbed on paraffin; slight glands in neck; *F. P. found: November 11th blood examined again; F. P. not found; not drowsy.*

(7) Danieri: Three months ill; glands in neck; no itching; typical aspect; from Kyagwe; F. P. found.

(8) Muyoujo: From Kyagwe; lived with No. 7; three months ill; glands in neck; itching; illness began same time as No. 7.

(9) Yesi: Illness began eight months ago; contracted in Bulemezi; body does not itch; glands in neck; staggers when he walks.

(10) Nsiko: three months ill; no glands; no itching; F. P. found.

* (11) Masitafa: In-patient; *F. P. not found after three examinations; undoubted case of sleeping sickness; died October 19th, 1901.*

(12) Susannah: Lives in Busi; taken ill last month; no itching; cervical glands enlarged; tongue tremulous; has special trembling at monthly periods; F. P. found.

(13) Mugatira: Taken ill six days ago, when film was taken; F. P. found; skin very irritable; cervical glands enlarged; complains chiefly of weakness; very tremulous.

(14) Wembuya: Has drowsy aspect; F. P. found; skin used to irritate; not now; glands in neck; tremulous.

(15) Erisa Makerere: Began to be drowsy nine days ago; fifteen F. P. found in one preparation; no glands in neck, except at angle of jaw; body irritable; tongue tremulous.

(16) —: Began to be drowsy a week ago; F. P. found; glands in neck enlarged; skin very coarse.

(17) Mikieri: Lives at Jungo; no glands in neck; skin not irritable; F. P. found; says he has greatly increased in drowsiness; blood tested a fortnight ago; no F. P. then found.

(18) Seruzi: Three months ill; tongue very tremulous; trembles as he walks; used to itch; does not now; glands in neck; F. P. found.

(19) Tabita: Tongue tremulous; ill one and a half years; pain in chest; F. P. found; body never itched; tongue and body tremulous.

(20) Mukasa: Has been ill one and a half years; F. P. found; glands in neck; skin irritable; not very tremulous.

(21) Balirwana: Has been ill five days; F. P. found; tremulous; skin not irritable; glands at left side of neck.

(22) Yudesi: Has been ill two months; F. P. found; had an attack of madness a few days ago; tongue very tremulous; skin irritable; small enlarged glands at left side of neck.

(23) Teretiwo: Was taken ill last month (October); F. P. found; tongue tremulous; skin not irritable; enlarged gland left side of neck.

(24) Mudu: Ill one month; F. P. found; lives in Bulemezi; tongue tremulous; skin irritable; glands under left jaw.

(25) Mukasa: Child; taken ill three days ago; tongue tremulous; no itching; distinct glands each side of neck; F. P. found.

(26) Musibika: Child; ill one month; tongue not tremulous; skin not irritable; glands in neck; F. P. found.

HÆMORRHAGIC PANCREATITIS IN ACUTE MALARIA.

By W. G. ROSS, M.D., and C. W. DANIELS, M.B.

London School of Tropical Medicine.

THE case, of which the history is related, presents several points of interest. There was hæmorrhagic pancreatitis and very extensive necrotic and other changes in the stomach and intestines, and yet pain was not a marked symptom in the course of the disease. There was nothing in the clinical history or in the symptoms, during the short period the patient was under observation, to indicate a malarial infection.

HISTORY OF CASE.

July 30.—P. S., aged 23. Fourth Officer Mercantile Marine, native of England, last voyage from Port Said, brought on stretcher from ship.

History from ship's surgeon: On 27th inst. surgeon thought he was looking ill when going about on deck, and sent him to bed; found he had slight pain and tenderness in the epigastrium. The same day hæmorrhage set in from the stomach and bowels, and recurred up to date of admission. The hæmorrhage was frequent and profuse. Patient had been on a run between Brindisi and Port Said, and was sent home to join another ship. Had not been ill from malaria. Had climbed up the mast to the truck about a fortnight before the attack, but had not apparently injured himself in any way.

Symptoms on admission, 2.15 p.m.—Temperature 102.8; pulse 140, small; skin cool and wet with perspiration, beads of sweat on face; is pale, but not anæmic, conjunctivæ, tongue, lips and finger nails good colour; tongue slight white coating; heart and lungs normal; liver area normal; spleen enlarged; felt just below costal margin neither hard nor diffuent. Abdomen: does not complain of pain, no tenderness, not tympanitic; no tumour discoverable. Urine 1020; acid clear, no albumen, no sugar.

5 p.m.: Vomited 8 ozs. of reddish-brown fluid, with a few dark clots. Shortly afterwards passed two stools of a similar character to the emesis, about the

same quantity, odourless, and containing no faecal matter. Ordered milk and lime water, iced. Pil. plumbi cum opio, four hourly.

8 p.m.: Temperature 98.4; is very thirsty; skin cold and clammy. Ordered Valentine's meat juice hourly; iced barley water.

10 p.m.: Temperature 97.4; pulse 120; stronger; skin not so cold. Has dozed a little, but wakes on being approached, and calls for drink.

July 31.—2 a.m.: Temperature 96.4; pulse 120; skin warm and moist; sleeping.

8 a.m.: Temperature 97.6; pulse 100, fuller and stronger; skin warm and moist; voice of fair strength. Has slept for the most part. Says he feels much better, but very weak from previous loss of blood. No tenderness of abdomen.

1.30 p.m.: Vomited 10 ozs. of fluid, reddish-brown colour, and a few clots, and in half an hour passed four stools of same character as emesis, and the quantity in all about 10 ozs. Was in great abdominal pain and distress; features pinched; respiration hurried; pulse 160; bathed in perspiration; abdomen tender; greatest tenderness between umbilicus and pubes. Hypo. morphine m. iv., atropine m. ii. given.

2 p.m.: Lips and finger nails cyanosed. Patient became unconscious; became rapidly collapsed, and died at 2.15 p.m.

Post mortem.—Six hours after death rigor mortis marked; body not cold; extensive staining (*post mortem*) on dependent parts; no signs of putrefaction; abdomen not distended; partial *post mortem* only allowed, all the viscera being removed through the abdominal incision. Body that of well-nourished, muscular young man. Moderate amount of subcutaneous fat; muscles a good colour, and in good condition; no excess of peritoneal fluid, and no hæmorrhage into cavity of peritoneum. There was blood-stained effusion under the peritoneum in the vicinity of the pancreas, at the base of the mesentery, and in small patches on various parts of the visceral peritoneum, but no actual hæmorrhages. The whole extent of the stomach and intestines was of a dusky-red colour, the intestinal walls were thin and flaccid, and the intestines not distended. The abdominal organs were removed *en bloc*. The pancreas was then seen to be of an intensely deep red colour—in sections almost black—not softened. On opening the stomach a considerable amount of blood-stained fluid gushed out, and the whole contents of the small and large intestines were of a similar character. The mucous membrane of the œsophagus was pale and normal in appearance. The whole internal surface of the stomach and small intestine was intensely congested. This congestion was greatest in the stomach and duodenum, and gradually diminished in intensity towards the ileum. The large intestine was similarly affected in a much smaller degree. The liver was enlarged, weight 5 lbs., soft and friable. The spleen, red in section, weighed 15 ozs., and was soft but not diffuent. The other abdominal viscera were healthy. The pleural and pericardial cavities could not be examined, but did not contain excess of fluid or blood clots; no adhesions. Lungs and heart perfectly healthy, and the latter contained

some recent red *post-mortem* clots. The gall-bladder was distended with very dark green bile. The brain was not examined. No fat necrosis noticed.

Microscopic examination of the pancreas showed numerous extravasations of blood; some of these, particularly those under the capsule, were extensive, but the greater number were small between the lobules, and sometimes separating the individual cells. In the portions examined neither cells nor fat was necrotic. The striking feature of the sections was the manner in which all the smaller capillaries were blocked with malarial parasites (*autumno-æstival*). Of these parasites, the majority were either sporulating or in a stage just before sporulation (*vide* drawings). The blood corpuscles in the larger vessels and in the extravasated blood did not contain any parasites.

The stomach and intestines showed a similar accumulation of parasites in the capillaries. In the mucosa, and to a small degree in the submucosa, there were numerous blood extravasations. The superficial layers of the mucosa were necrotic, and had been invaded by micro-organisms of several species, but a streptococcus was the most abundant and most constantly met with.

In the spleen, liver, and kidneys, and lymphatic glands, an occasional malarial parasite was found after a prolonged search, but in no part was there any accumulation of parasites similar to that met with in the pancreas and intestines. There was no evidence of any but quite recent malaria in any of the organs.

No micro-organisms, other than malaria parasites, were found in any organ but the alimentary canal. There was little evidence of hæmolysis, but there were a few yellow granules in the hepatic cells and in the spleen; there were a few granules giving the reactions of loosely-combined iron.

The probable sequence of events is as follows:—An infection with malaria took place at Brindisi. During the voyage home the malaria remained latent, or at any rate, the parasites were not in sufficient number to cause marked symptoms of "fever." At each sporulation they increased in numbers, and in this case sporulation took place in the capillaries of the intestines and pancreas. Local blood stasis, due to the blocking of the capillaries in these regions by the parasites, was produced, and caused sufficient damage to the mucosa to allow the micro-organisms present in the intestinal contents to invade it. A superficial necrosis of the mucosa resulted from the effects of the bacteria on the congested intestinal walls.

Whether the pancreatitis was directly due to the malaria parasites, or of secondary bacterial origin, it is impossible to ascertain.

Cases of hæmorrhage into the alimentary canal have been recorded by many observers as a rare complication of malaria. Hæmatemesis, melæna, or motions of a more dysenteric character, have been observed. Recovery is the rule, but in fatal cases the capillaries have been found—as in this case—blocked with corpuscles containing malarial parasites. In some of the cases, collapse has been the prominent feature. In one such case under my own

(C. W. D.) observation, there was both hæmatemesis and passage of blood by rectum, associated with profound collapse. This hæmorrhage did not recur, as quinine was given hypodermically, but I (C. W. D.) am informed that subsequently there was methæmoglobinuria, and the case terminated fatally from suppression of urine.

The causation of the algid forms of malaria is probably closely connected with an accumulation of the parasites of malaria in one of the abdominal viscera instead of in the commoner sites, the brain or lungs.

The main points of interest in this case are:—

- (1) The unusual site selected for the sporulation of the parasites, viz., the pancreas.
- (2) The hæmorrhagic pancreatitis associated with the condition.
- (3) The absence of any previous symptoms of malaria.
- (4) The extensive invasion of the alimentary tract by the malaria parasites, and the secondary necrosis of the mucosa, though this must have been mainly due to the bacterial invasion.

MARGINAL ULCERATION OF THE GUMS OCCURRING AMONG NATIVES OF EAST CENTRAL AFRICA.

By NEIL MACVICAR, M.B., C.M.

Late Medical Officer to the Blantyre Mission, British Central Africa.

THIS disease (native name *chikusa*), is common among the natives of the Shire Highlands. It is an unpleasant and painful malady, though so far as I have seen it is not dangerous to life. I have not seen it anywhere described.

The ulceration commences at one spot on the alveolar margin of the gum, commonly in front, and in a few days it has spread a couple of inches or more along the gum. In advanced cases the narrow line of ulceration may extend continuously the whole length of both upper and lower jaws, from one side of the mouth to the other. It never—or very rarely—extends outwards further than a quarter of an inch from the teeth.

The ulcerated surface presents an eaten-out appearance, discharges dirty-looking pus, and is speckled with blood clots, indicating the mouths of bleeding arterioles. There may be a good deal of bleeding. The ulceration eats in so as to expose the roots of the teeth, which, in consequence, become very loose. Sordes collect on the teeth close to the ulcer. The gums are swollen and purplish in colour. The breath is offensive. The tongue is usually only slightly coated. There is no accompanying sore throat.

The disease lasts for weeks, though how it would end if untreated I do not know. If at all extensive, it prevents the sufferer from eating solid food.

The cause of this disease is, I should think, a specific micro-organism, though what its nature may be, or in what way it gains access to the gums, I have not been able to investigate. I have not noticed its occurrence in different members of the same family, or other evidence of its being transmitted from

person to person. It occurs at all ages, except among infants. The worst case I have seen was that of a child, aged 4, whose teeth seemed almost to be dropping out, and whose whole face was so much swollen that the mouth could hardly be opened. Many of the patients were well-nourished young men and women, who, moreover, were habitually careful about the cleansing of their teeth after meals.

Treatment.—After experimenting with various antiseptic mouth washes, I found that by far the most effective was a moderately strong solution of permanganate of potash. This, if used frequently—half a dozen times a day, or oftener in bad cases—will in a few days cure even the worst cases. When, owing to the swelling, it is difficult for the patient to move the lotion about in his mouth, it can be applied by means of a camel's hair brush gently pushed along between the gum and the cheek. Under this treatment the fœtor rapidly disappears, and the edge of the ulcer grows in towards the teeth. Even after complete cure, however, I have seen the roots of the teeth remaining exposed owing to the loss of tissue.

NOTE ON HÆMORRHAGIC BULLA OF THE MOUTH.

I was much interested in Dr. Preston Maxwell's account (*JOURNAL OF TROPICAL MEDICINE*, July 1st, 1901) of this condition as it occurs in China, because I had myself met with a case in Central Africa similar to the ones he describes.

The patient was a young man, a native of the Katanga district of the Congo Free State. The bullæ had appeared, he said, some weeks before I saw him, while he was passing through Mpeseni's country on his way to Blantyre.

I saw a black bulla covering an area about the size of a threepenny piece on the dorsum of the tongue, just to the left of the middle line. Three smaller ones were seen on the inner aspect of the lower lip. On the following day the bullæ had burst, and there were clots of blood in the mouth. Two days later the places had almost healed.

After reading Dr. Maxwell's account of his cases, I am inclined to think that I may have misunderstood the patient when I took him to say that the bullæ had been there for so long a time. He may have been referring to the onset of certain other symptoms, apparently due to a mild attack of dysentery, from which he appeared to be suffering.

This is the only case of the kind I have seen, and the bullæ were much smaller than those described by Dr. Maxwell. The case, however, appears to be of a similar nature to his, and as its occurrence in Africa may be of interest to investigators, I have ventured to publish this note.

BACTERIOLOGICAL LABORATORY IN TURKEY.—The Sultan has sanctioned the erection of a bacteriological laboratory at Silvi-Bouroun, an isolated spot on the Bosphorus, near Beicos. Huts for plague cases are already in existence there. The Turkish Government has sent Drs. Chevki Bey and Nilrath Bey to India to study plague.

MALARIAL FEVER AS MET WITH IN THE GREAT LAKE REGION OF CENTRAL AFRICA.

By ALBERT RUSKIN COOK, M.D., B.Sc.Lond., B.A.Camb.
Late Scholar of Trinity College, Cambridge.

(Continued from p. 43.)

MOTILE BODIES.

THESE are of two kinds; the first consists of very small pigmented bodies possessed of most active movements and most distinctly locomotive. Their motion is quite distinct from Brownian movement. For this reason they are probably not broken-off fragments of adult plasmodia exuded through pressure on the swollen blood corpuscle. They probably represent some kind of motile microgamete. Care should be taken not to confound their motion with that of granular *débris* set up by currents caused by varying degrees of pressure on the cover-slip.

The second kind is the well-known flagellated body. Out of a consecutive series of over 200 cases I only observed it six times (or rather in six cases) in blood

the plasma, and when it encounters a red blood corpuscle generally attaches itself by one extremity to it. Sometimes, as if dissatisfied with its reception, it breaks off again and once more locomotes, apparently seeking for its macrogamete.

More rarely the flagella may be seen attached to their parent parasite as in Case 151, fig. 4. Usually, however, what is noticed is one or two flagella projecting from the periphery of a red blood corpuscle. In fresh specimens the whip-like movements of the flagellum may violently agitate the corpuscle. In stained specimens the flagella seem to spring from the extreme periphery of the corpuscle; see fig. 4, Case 106, which are copied drawings of specimens under a magnifying power of 1100 (Oc. 3, 0.1 $\frac{1}{8}$ lengthened tube).

I only noticed the presence of crescents in three out of over 200 cases, and even these I was not certain of. At first I put this down to faulty technique, but continued observation showed the same anomaly, and therefore I was interested to read in a paper by Plehn (Physician of the German Government in Cameroons), translated in the JOURNAL OF TROPICAL MEDICINE,

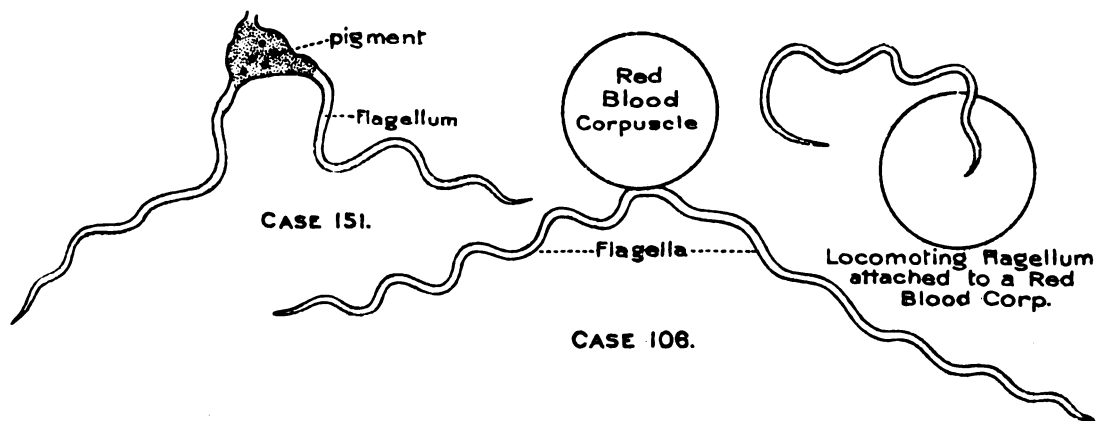


FIG. 4.

drawn direct from the body and examined at once. The statement in the ninth edition (1900) of Green's "Pathology," that "flagellated organisms are never found until the blood has been withdrawn for about a quarter of an hour" is quite incorrect. The very first case in which I saw it (not one of the series mentioned above), that of an anæmic and emaciated child, on making a fresh specimen and examining at once, the whole field seemed to be full of a mass of waving and lashing flagella coming from nearly every corpuscle. It reminded me of the rippling effect produced by the wind passing over a cornfield. The child was desperately ill and died the same night. Careful examination in similar cases has failed to detect any "crescent" bodies which could have been transformed into the flagellated bodies. On the other hand, the flagella generally seem to spring in pairs from unaltered red blood corpuscles. However, this may be merely due to the curious adhesive property of the free flagella.

Nothing is more interesting than to watch the progress of a free flagellum; it makes its way through

January, 1900: "The half-moon forms (crescents) of the malarial parasite which alone are supposed to be capable of transmission are in Cameroon extremely rare; in fact they occur quite isolated and are not to be found during many of the worst fever months. I saw, during almost four years in Cameroon, but once flagellated forms." And again, in a paper by Dr. Hans Ziemann, Staff Surgeon, Victoria: "I thought with longing and secret envy of the material at the disposal of the Italian investigators whose patients often had masses of crescents in their blood. Out of more than 1000 examinations of blood I could only detect on twelve occasions isolated crescents" (translated in the JOURNAL OF TROPICAL MEDICINE, January, 1901). Probably the immediate ancestor of the flagellated body is a spherical, and not a crescent-shaped body.

KARYOCHROMATOPHILES

Karyochromatophiles, or primitive bodies, was the name given by Plehn to certain very small bodies which eagerly absorbed nuclear stains (JOURNAL OF TROPICAL MEDICINE, December, 1900, and January,

1901), and which he regarded as the cause of the severe afebrile anæmia met with in the tropics, and the precursors of the plasmodia, enabling the latter to exist during the long latent periods during which no febrile manifestations are seen, but which on suitable provocation, *e.g.*, chills, over-fatigue, &c., are terminated by an explosion of fever. This view has much to recommend it, and I have on several occasions noticed very small granules, stained by methylene blue, in red blood corpuscles in people liable to fever, though not actually in an attack. More evidence is needed, however, as to their presence and development; I believe I have seen all stages between this form, which may be regarded as the smallest form of a plasmodium, and the adult parasite.

PRESENCE OF FREE MELANIN IN THE BLOOD.

Davidson states (p. 114) that "for purposes of classification, the presence or absence of melanæmia may be taken as pathognomonic of malarial fever." Other observers have pointed out that this is not strictly true, melanin being found free in other diseases. Here I would only lay stress on a fact that I do not remember seeing mentioned by anyone, *viz.*, that in the blood of new-born infants, and for some little time after their birth, granules of hæmoglobin or pigment, indistinguishable from that manufactured by the parasites, may be found in considerable quantity, both free and intracellular. Possibly this is connected with the icterus neonatorum.

LEUCOCYTES IN MALARIAL BLOOD.

The various proportions of the different varieties of leucocytes met with in fever, and the changes they undergo during the chill, hot and sweating stages is a most interesting subject, and one my attention was directed to by reading the Report of the Government Commission on Fever in Nyassaland. I have no definite statistics to offer on this point, but it is one I hope to carefully examine later on. Meanwhile the impression left on me by a large number of observations is as follows: Marked leucocytosis in fever is rare, and probably never occurs without some secondary complication, *e.g.*, pneumonia. I only remember seeing it two or three times in my series of cases. As a rule, the lymphocytes are not largely increased in number. There is, however, probably an increase in the finely granular oxyphile (polymerous nuclear) cells, this latter is the chief phagocytic agent as regards the parasites, I think the number present of this latter kind of leucocyte may be of value to a certain extent in forming a prognosis. If they are numerous the reacting power of the patient is probably good, and phagocytosis will make an end of his parasites. In mild cases of fever the attack often rapidly ceases by an auto-curative action. Eosinophilous cells are not apparently increased in number.

MORBID ANATOMY.

Here I have nothing fresh to add to the well-known facts recorded by others. The enlarged and pulpy spleen, the softened and frequently enlarged liver, and the deep melanosis exhibited by both these organs are very striking objects. I should like to lay emphasis on the frequency with which adhesions are found, especially on the diaphragmatic surfaces of both

spleen and liver. There can be no doubt that the congestion of these organs, always present to a greater or less extent, lends itself very readily to an inflammation of the covering peritoneum and accounts for the severe pain so often complained of during the course of the attack. In the comparatively small number of *post-mortem* examinations I have been permitted to make by the relatives of the cadavers, I have not noticed any changes in the kidneys. Smears taken from spleen and liver usually show the presence of parasites, but by no means abundantly so, if the patient has been well cinchonised before death.

INCUBATION.

From the practical side this is of comparatively slight importance. I suppose one never sees a native in a primary attack, unless indeed two babies, aged 4 and 5 days respectively, in whose blood I found the plasmodia and who presented the clinical features of fever, were instances of such. Europeans as a rule enjoy immunity for the first few months of residence, but the opportunities of infection are so numerous, and the *Anopheles* mosquito so ubiquitous, that it is exceedingly difficult to judge. I have often noted, however, that first attacks come a few days after crossing the Lake, the papyrus-fringed shores of which are grand breeding places for mosquitoes. The first attack seems to be usually a benign tertian, but on at least two occasions I have seen a rather severe irregular remittent yielding only very slowly to quinine. Once fever has manifested itself many different causes will bring about a recurrence within twenty-four hours of the exciting cause, *e.g.*, a chill, over-fatigue, undue exposure to the sun, &c.

GENERAL SYMPTOMS.

Pursuant to the course described above I shall not attempt to describe the classical symptoms of the intermittent form. In the rapidity of the onset, the rigor, cold, hot and sweating stages, they exactly correspond to the well-known descriptions given in all the text-books. This form is distinctly uncommon amongst natives, and not very common amongst Europeans. Proceeding then at once to the ordinary remittent type of fever which forms the immense bulk of all cases seen in our hospital practice, I find that out of over 400 cases, the mode of origin, whether by rigor or not, is noted in 109 cases. Of these 59, or 54 per cent., started with a rigor; as the native expression for this merely means "to shake," probably chilly feelings were often felt in the remaining 46 per cent. Personal experience confirms the fact that rigors are an exception, sensations of cold or chilliness being usually felt. Yawning and stretching of the limbs frequently herald an attack, though at the time the thermometer may register a normal temperature. Severe prefebrile headache is uncommon, though a dull aching headache may be complained of. Diarrhoea, contrary to what is often stated in the text-books, is an exceedingly frequent concomitant. Out of 121 cases in which this condition was inquired into, the high proportion of 35 per cent. (43 cases) was found in which this condition heralded an attack.

One gentleman possessed of great observing powers told me he always knew when he was in for an attack

of fever by the smarting sensation caused by micturition, a premonitory sign he found to be infallible. Very heavy sleep, almost amounting to torpor, has been noted by some as one of the earliest symptoms, or strange to say on the first day of actual fever.

The skin is usually dry; a good sweating at the remission is distinctly favourable. More often only a slight moisture is perceived. The conjunctivæ are often inflamed and severe photophobia may be present. This is often well marked in Europeans. Jaundice is best recognised in the conjunctivæ of the natives; it is only common in the bilious remittent type and in black-water fever. In 232 cases, where this complication was specially looked for, it was only noted in 10 cases (4 per cent.), of which, however, three died. Severe jaundice is certainly a serious complication, slight jaundice is of no moment.

The pupils are usually moderately dilated.

The tongue is heavily coated with a white fur in the early stages, later on it becomes dry, brown, and markedly tremulous as the patient passes into a typhoid condition. This latter condition is, however, rare, unless the patient has been for long untreated before being brought to the hospital. In this case the teeth become covered with sordes, and subsultus tendinum or carphology may be present, with a low, muttering delirium.

Anæmia is always present and may be very marked, even after one attack. It is more fully referred to under the head of complications.

PYREXIA.

In the commonest form of African fever met with in Uganda, the temperature shows an irregular remittent curve. Often this shows tertian exacerbations. It is frequently very resistant to quinine, the temperature taking from four to ten days to reach the normal line.

Out of 227 temperature charts 178 (79 per cent.) were found to possess this kind of curve. The pyrexia is of the following types:—

- (1) Simple tertian intermittent. Common.
- (2) Double tertian intermittent (quotidian). Not very common.
- (3) Quartan intermittent. Rare.
- (4) Remittent tertian. Very common.
- (5) Remittent quotidian. Not very common.
- (6) A quite irregular chart. Very common.

Experience shows that the last is most difficult to deal with as it is often very resistant to the action of quinine. Associated with the first three types are the large pigmented plasmodia which sporulate in the peripheral blood. Associated with the last three are the much smaller less pigmented parasites. The asexual forms only sporulate sparsely in the peripheral circulation. The histology of the parasite has been described already. In the last type the behaviour of the temperature to quinine may show a regular drop from evening to evening till the normal line is reached. In less malignant attacks a resolution by crisis may take place. I have again and again seen the temperature drop 7·8, or nearly 10 degrees, in one night after a single large of quinine. In these cases, even if the quinine be discontinued, the temperature does not rise again.

A very common complaint is that of fugacious pains, which may be so severe at times as to evoke cries of distress from the sufferer. I have had several times to give morphia hypodermically for this. The seat of the pain seems to be most frequently in the back, loins, or the bones of the legs or arms. It is probably due to a slight neuritis, and seems to have no connection with the kidneys. Quite distinct from this is præcordial distress due to over-action of the heart, especially when the patient is walking about when he should be in bed, and the great tenderness often complained of in the right hypochondriac, epigastric and left hypochondriac regions; the pain in the first two regions is due to a congested and tender liver, in the last to an engorged spleen or to perisplenitis.

In remittent fever the pulse which is at first hard and full, has a very forcible beat, which may cause complaints of a throbbing headache from the patient; later on it becomes progressively softer, weaker, and more dicrotic, finally becoming rapid, small, and irregular, with very low tension. I have been sometimes surprised, however, to notice how regular and comparatively strong the pulse has been within a few minutes of death.

THE RESPIRATION.

The respiration is only hurried in proportion to the height of the temperature. Of course co-existent splenitis, pneumonia, or bronchitis may cause profound alteration in the respiration rate. With the exception of initial diarrhœa, which, as has been pointed out, is very common, the bowels tend to be constipated throughout. Profuse diarrhœa would make me suspicious of typhoid or dysentery. A watery diarrhœa with collapse, the so-called algid symptom in pernicious fever, is not uncommon, especially in infants. It yields very readily to quinine in full doses, with some astringent added. Herpes, though met with, is uncommon. Initial epistaxis is common. This symptom is also met with in malarial cachectics suffering from enlarged spleens. The malarial ulcers described so graphically by Dr. Cross in the JOURNAL OF TROPICAL MEDICINE, November, 1900, I have never seen, though I have seen thousands of cases of ulcers in natives, and many of these doubtless have malarial attacks from time to time, which impedes the natural function of repair. Some of the appearances he describes seem to me to be characteristic of oriental sore. Malarial scurvy as described in cases occurring in India I have never come across, though phagedæna of the gums in syphilitic cases and fever may occur together.

As regards cerebral symptoms these are met with in all degrees, from the dull, throbbing headache to the fulminating attacks of coma which may kill a strong man in a few hours. Delirium is common, especially at night. Active maniacal delirium is fortunately rare. Cerebral irritation is frequently met with in the graver types of irregular remittent fever. The patient lies in a condition of general flexion on his side, and if aroused answers irritably, if at all. He refuses food and medicine, and violently resents changes of posture or attempts to open his closed lids.

Coma is a serious though by no means fatal condi-

tion. A patient with wide-open, staring eyes, fixed pupils, and stertorous breathing, quite incapable of understanding anything said to him, may yet recover rapidly under the influence of a large dose of quinine administered hypodermically.

In the bilious remittent form the patient vomits freely a quantity of green bile, and is usually deeply jaundiced, as shown by the deep yellow conjunctivæ and ochreous skin (if a European), pale stools and presence of bile colouring matters in the urine. This form of fever is dangerous unless taken in hand early.

On reviewing the clinical types presented by several hundred cases one would arrange them thus :—

- (1) Intermittent fever.
- (2) Remittent.
 - a. Mild
 - b. Severe { Gastric type
Bilious remittent.
 - c. Pernicious { Comatose
Dysenteric
Syncopal
Adynamic
Algid
Hyperpyrexial.

(3) Hæmoglobinuric.

The chief characteristic of each type is sufficiently shown by the title. The commonest forms of pernicious fever in Uganda are the comatose, syncopal and dysenteric.

The vexed question as to whether hæmoglobinuric fever, or black-water fever as it is universally called here, should be included among the malarial fevers, may be briefly discussed. The floods of papers that have appeared on the subject cannot be said so far to have definitely settled it. A few preliminary statements may first be given to clear the ground. As met with out here, it is not due, with all due regard to R. Koch, to the abuse of quinine.

Though rarely met with amongst natives I have yet met with two cases of it amongst them. They are said to suffer less than Europeans when attacked, and my two cases both bear this out.

It differs from malarial fever in one notable point. Quinine has not a specific influence upon it. Ordinary attacks of malarial fever seem always to precede it. Though commonest in the second or third year of residence in the country, it may be met with for the first time in a person who has been in Africa for many years. Thus one Englishman fell ill of it here in his twelfth year of residence. When it has once attacked a person, he is very liable to relapses.

Space only permits a very brief description. The typical attack is ushered in by premonitory feelings of malaise; a single prolonged rigor forces the patient to bed, if he is not there already, and with shaking hands he heaps the blankets over him. A sensation of burning heat follows, with, it may be, sharp pains in the loins. If a stranger to this fever, on passing water he will be startled to see it the colour of porter, or it may be blood-red or like port wine. Jaundice and most distressing vomiting quickly set in, everything being rejected, with usually bilious vomiting. The secretion of the kidneys gets much less, and intractable insomnia is usually a marked feature. Severe anæmia rapidly develops and a loud hæmic murmur may be heard all over the cardiac area.

Muscular prostration becomes intense, the pulse flags, and an anxious expression becomes stamped on the countenance. At this stage improvement may set in and the patient recover, the urine becoming more abundant, the vomiting ceasing, and the jaundice diminishing. He will remain weak and anæmic for long, however; or a fatal issue may rapidly supervene. The urine may become clear again before the last, or suppression may occur, only a few drops being drawn off by the catheter, and these becoming solid on boiling. Repeated rigors may occur. Sudden syncope may abruptly cut the thread of life short.

Our European who had had several attacks told me he could always tell when he was passing black-water urine, even in the dark, by the noiseless manner in which micturition took place in the chamber (probably due to the highly albuminous character of the urine).

As regards the presence of parasites in the blood it must be remembered that the disease occurs generally amongst Europeans, that one is usually summoned long distances to see the patient, and on arrival he is pretty well cinchonised. In one case in which I got some good preparations of blood I found no parasites; but the patient had taken large doses of quinine.

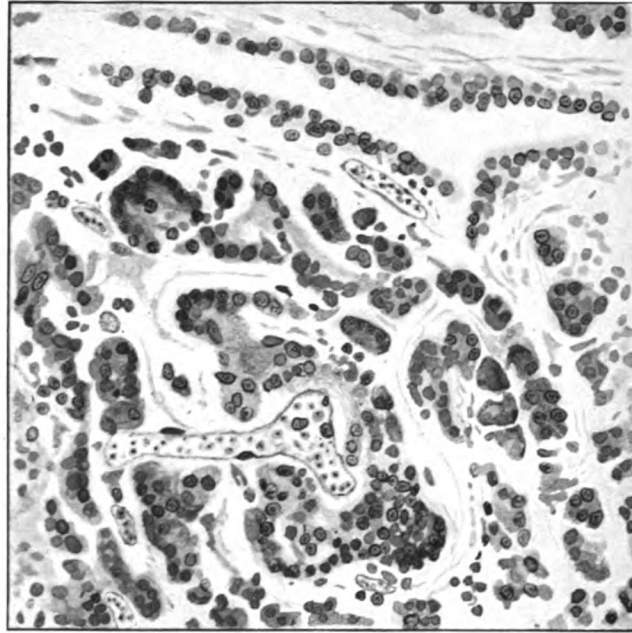
In a great many cases of fever the spleen may easily be felt below the ribs in the left hypochondrium. The large notch in its anterior surface, its movements with respiration, the dull percussion note over it when large enough to approach the abdominal wall, all serve to verify it. In slight attacks it covers its former volume almost completely after the fever has passed away, and it is interesting to follow with the hand its diminishing size, day by day, till it becomes lost once more beneath the ribs. In frequently occurring attacks a species of hyperplasia sets in and the organ remains permanently enlarged. In some cases this enlargement is immense, the organ filling two-thirds of the abdominal cavity and occupying the epigastric, left hypochondric, left lumbar, left iliac, hypogastric, umbilical, and right iliac regions. In these cases it may feel almost of stony hardness, and may be quite insensitive to pressure. In some cases it is associated with ascites. Out of 280 cases in which the condition of this organ was specially noted, it was found enlarged in 127 (45·3 per cent.). Of course an enlargement sufficient to be felt below the ribs must be very considerable.

Similarly the liver was felt to be enlarged in 17 cases out of 235, a low percentage of 7·2. Congestion of the liver not leading to any enlargement that can be detected by palpation was very common.

As regards albuminuria in malarial fever, out of 91 cases in which albumin was tested for, it was found in 66 cases (72·5 per cent.). This includes cases in which only a slight amount was detected. A large amount was rare, but did not add materially to the gravity of the prognosis.

RECURRENCES OR RELAPSES.

These are very frequent. In untreated cases in which the fever spontaneously subsides, attacks are very liable to recur about a week from the subsidence of the last attack. In women, the catamenia is a time in which attacks are very common, and some European ladies seem to get an attack every monthly period.



I.—SECTION OF PANCREAS, SHOWING CAPILLARIES FULL OF PARASITES.

In the upper part is a pancreatic duct.

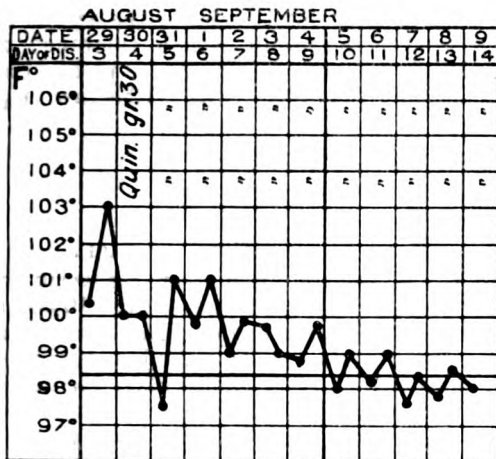


II.—OBLIQUE LONGITUDINAL SECTION OF SINGLE CAPILLARY, SHOWING PARASITES IN VARIOUS STAGES.

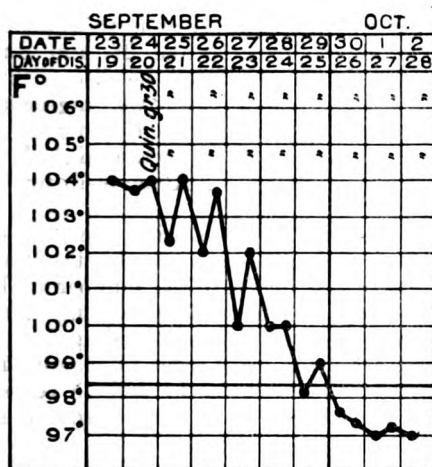
Enlarged from No. 1.

To illustrate the paper on "Hæmorrhagic Pancreatitis in Acute Malaria," by W. G. ROSS, M.D., and C. W. DANIELS, M.B.,
London School of Tropical Medicine.

Another interesting point in connection with the more obstinate infections, and one that I have observed over and over again, is that in a patient whose temperature has dropped to normal under the influence of large doses of quinine, fresh attacks may take place, being ushered in by a rigor and sudden rise of temperature.



CHART, SHOWING GRADUAL REDUCTION OF TEMPERATURE UNDER QUININE.



A SIMILAR CHART.

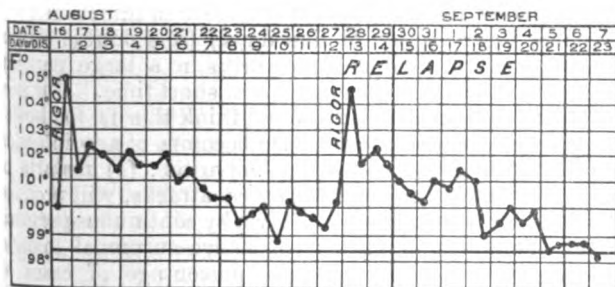


CHART OF BLACKWATER FEVER.

I have found that after a severe attack has been cured, prophylactic doses of ten grains of quinine daily

are not sufficient in all cases to prevent relapses; nothing is more disappointing in such cases than to see after the lapse of seven to ten days the temperature run up again and a rigor take place. If, however, the patient has been free of fever for some weeks, much smaller doses, three to five grains, are sufficient to prevent recurrences. When once a person has become, as it were, saturated with the malarial poison, any little cause that lowers his vitality may cause a relapse, though the prompt use of quinine may ward such off. The commonest cause is a chill either from sitting in a draught when perspiring, or from getting wet feet, or exposure to a strong wind; next to this, perhaps, comes over-fatigue, the presence of another illness, undue exposure to the sun, &c.

Traumatic injuries are not nearly so likely to bring on an attack of fever. In treating upwards of 150 cases of malarial disease breaking out amongst them as a direct consequence of the injury. Quinine taken even during the premonitory symptoms of an attack seems often to abort the succeeding rise of temperature.

COURSE AND TERMINATION.

The usual result of an attack of fever when taken early and suitably treated with quinine, is prompt defervescence of the fever and rapid convalescence. Remittent fever yields less readily than intermittent, but it is surprising how often even a chronic infection yields to quinine, the temperature curve steadily sinking day by day, till in three to five days the normal line has been reached. Occasionally, however, even when quinine is given early and in adequate doses, the temperature keeps up for an unduly long time.

FATAL TERMINATION.

Cases coming early under treatment very rarely fall into a typhoid state. Pernicious symptoms may spring up, a comatose or algid state may supervene, or the life may be cut short by syncope or pneumonia, or other serious complications, but in my experience the illness seldom becomes chronic.

One arrived in Africa with an *a priori* belief that malarial fever was a comparatively harmless disease to natives, and that death seldom ensued from it. Accurate statistics completely negative this pleasant idea. From May, 1897, to July, 1901, 456 cases of fever were admitted into the Mengo Hospital, of which 54 died, giving a mortality of 11.8 per cent. Of course some were brought in in a moribund condition and others practically starved from suitable food, but even subtracting 18 deaths which occurred within forty-eight hours of admission, the corrected death-rate stands at 8 per cent. Very different is this to the assertion made so lightly that fever is a comparatively innocuous disease to the native of Africa. Amongst these 53 deaths I find the following immediate causes are represented:—

| | No. of Cases. | | No. of Cases. |
|----------------------|---------------|------------------------------|---------------|
| Syncope and collapse | .. 5 | Anæmia and marasmus | .. 3 |
| Meningitis | .. 4 | Intense plasmodial infection | 3 |
| Coma | .. 4 | Delirium | 2 |
| Associated typhoid | .. 4 | Gangrene | 1 |
| Jaundice | .. 4 | Epilepsy | 1 |
| Dysentery | .. 4 | Bronchitis | 1 |

DIAGNOSIS.

This has been much simplified by discovery of the plasmodium malarie. If definitely found, whatever else may be there, malarial infection is certainly present. Conversely, if repeated examinations show its absence it may be confidently excluded from the diagnosis.

It must, however, be remembered that the parasites may be found in the blood even in considerable numbers without any clinical manifestations of fever. I have many times found them in the blood of young native children who exhibited none of the usual signs of fever. Also merely finding the plasmodium does not necessarily prove that the patient is not suffering from a mixed infection. Nor is this wonderful when we remember how prone any disturbance is to light up any old malarial infection. I have found the parasite of malarial fever in the blood in cases of typhoid, pneumonia, small-pox, bronchitis, filariasis, sleeping sickness, appendicitis, and dysentery, thus showing a double or mixed infection.

TYPHOID FEVER.

The first four years of my stay in Uganda, though suspecting the presence of typhoid fever in the country I could not definitely prove its existence. The reason of this, of course, lay in the extreme difficulty in a semi-civilised country of obtaining permission for *post-mortem* examination, the natives accusing us of wishing to eat the heart, &c., of the cadaver. However, tact and increasing intelligence in the people are gradually leading to a much better state of affairs. Another difficulty has been the demands of an exceedingly busy hospital practice on a very slender staff, so that it not unfrequently happened that when the looked for opportunity came lack of time rendered only a partial examination possible.

No other medical men whom I met up here could give me positive information, and the Government P.M.O. only a few months ago told me that he had never come across a case. From time to time we had cases in the hospital whose clinical course resembled that of typhoid, but as unfortunately at present Widal's serum test had been quite impracticable, one could not be positive. In the light of subsequent discovery it was seen that there must have been a fair number of cases of typhoid, though the temperature charts in some were modified by co-existent malaria.

On February 21st, 1901, a woman was admitted suffering from malarial infection, as proved by the detection of the parasites on a blood examination. In spite of the usual treatment she got worse, and miscarried on February 23rd; on February 25th acute peritonitis set in, with intense tympanitis, &c. I did a laparotomy and found signs of commencing peritonitis with thick lymph on one coil, but did not notice any perforation. Much purulent fluid was got rid of by repeated flushings of the peritoneal cavity and the abdomen sewn up. The patient died two days after.

On April 2nd, 1901, a man was admitted, greatly exhausted, with a high temperature. A film of blood showed an intense infection of large pigmented plasmodia belonging to the tertian variety; the type of fever was quotidian. Quinine was freely given, and

blood preparations showed the rapid diminution of the parasites. His temperature continued high, however, and on April 11th he died. Of course he had arrived in a very bad condition from want of food. Exigencies of work only allowed a partial *post mortem*. Smears were taken from the liver and spleen, and it was noticed that the mesenteric glands were much enlarged.

On examining the smears, besides the usual appearances of a case of fatal malaria, the spleen smear was crowded with bacilli, which examined under $\frac{1}{2}$ in. oil immersion and No. 5 eye-piece appeared morphologically identical with Eberth's typhoid bacillus.

(To be continued.)

PLAGUE.

PREVALENCE OF THE DISEASE.

INDIA.—The plague returns for the week ending January 18th show, 9,204 deaths against 10,368 in the week preceding. The principal figures are: Bombay districts, 3,510 against 4,797; Punjab, 2,379 against 2,662; Mysore State, 653 against 636; Madras Presidency, 590 against 586; North-West Provinces and Oudh, 829 against 685; Bengal, 593 against 462; Kashmir (Jammu), 257 against 192. In Bombay city, during the weeks ending January 11th and 18th, the plague deaths numbered 234 and 296 respectively.

EGYPT.—During the weeks ending January 25th and February 1st, the number of fresh cases of plague reported in Egypt were 15 and 22, and during the same periods the deaths from the disease numbered 16 in each week. The disease is well-nigh confined to the city of Tintah, but cases have also been reported at Aboukir in the Mehalla district, at Kafr Enan, near Zifteh, and at the village of Koum-el-Nour, near Mit Ghamr.

CAPE OF GOOD HOPE.—During the weeks ending January 11th and 18th respectively, the number of fresh plague cases in the Cape numbered 3 and 2. There were no deaths from plague during either week.

MAURITIUS.—During the weeks ending January 30th and February 6th, the fresh cases of plague in Mauritius reported amounted to 12 and 13 respectively; during the same periods the deaths from the disease numbered 9 and 8 respectively.

NEPHRITIS IN MALARIA.—Moore gives the conclusions of his paper in the following: (1) Nephritis is not likely to occur in a single tertian infection for a short interval, say of five days; (2) a double tertian infection will produce a nephritis in a large percentage of cases if it runs only for a short time. Our percentage is 80 per cent., but I think this is too large; (3) the more chronic the case becomes of any infection the more likely to produce nephritis; (4) malaria of long duration, or often repeated attacks, will produce chronic renal disease, as shown by continuous presence of albumin and casts; (5) æstivo-autumnal malaria probably gives the greatest percentage of cases of nephritis—68.7 per cent; (6) the age of the patient, height of temperature, or specific gravity of the urine showed no relation to the presence of albumin and casts in our cases.—*American Medicine*, Philadelphia, December 28th, 1901.

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THE

Journal of Tropical Medicine

FEBRUARY 15, 1902.

THE MEDICAL SERVICE OF THE ROYAL NAVY.

At the present moment, when radical changes are being introduced in the medical services of the Army, Militia, and Volunteers, and also in the Colonial Medical Service, we are not without apprehension that the medical service of the Navy may be neglected. We have frequently expressed regret that the surgeons of the Navy contributed so little to the medical journals, but this, as we have explained previously, is owing not to want of ability or interest in their work, but to the cumbrous nature of the means of transmission the medical, in common with all, officers of the Navy are subjected to. We have received from time to time, from surgeons of the Royal Navy, important and valuable papers, forwarded by the courtesy of the Director-General. We should like to see our professional brethren

in the Navy freed of this roundabout way of communicating their observations, and placed on the same footing as, say, the officers in the Indian Medical Service. The surgeons of the British Navy have unique opportunities of observing disease in many climates, and as pioneers and scouts in the van of tropical medicine their experiences would be of immense value to medicine. We hope to see this incubus removed at no distant date, and we are sure that the authorities at the Admiralty, were the matter placed properly before them, would grant the permission we suggest.

For yet another reason would we wish to see more freedom given them to express their opinions. The medical officers of the Navy have been in much the same position as regards position, leave, pay and allowances, for many decades, and it is impossible for naval officers to raise their voices in their own behalf. The discipline of the Service is such that a murmur of discontent is almost unknown.

The medical service of the Navy is not composed of "sea lawyers" by any means; on the contrary, the medical officers of the Navy err, if they err at all, by being but too well satisfied with what, to the outside members of the profession, appears unsatisfactory in many respects. They are loyal to their Service and jealous of its reputation; but their silence on many occasions—compulsory it may be—is not always to the best interests of the Navy.

The sister medical service in the Army is ever on the *qui vive* to better its position and to improve the hygienic conditions of the soldier. Better terms are from time to time bestowed on the military surgeon, in consequence of a form of agitation which in the Navy is impossible. This is not quite as it should be, and unless the Admiralty bestir themselves, they may find their Medical Department neglected and the services of medical men difficult to obtain. We hear a scheme is being prepared by the Admiralty to redress the various "grievances" of the Medical Department of the Navy. Unless this is done effectually and considerately, the Naval Medical Service will fall behind in its competition with

the R.A.M.C., with the result that it will not only be difficult to secure good men for the Service, but even to secure medical men for the Navy at all.

The grievances, if they may be so designated, are several: (1) The present rate of pay is relatively below what is granted to the R.A.M.C. officers, and is less amongst newly-joined officers than is now-a-days earned by recently-qualified medical men in civil practice. (2) The pension of £1 a day would appear satisfactory, but instead of the increments of pension being obtainable only after three or four years' periods of additional service, it would be better were the pension increased by one shilling a day for each year's service after the twenty years' service has been completed. (3) Sending a fleet-surgeon, after twenty-five or more years' service, to sea in any but a flag-ship, would appear to be not only inexpedient but wholly unnecessary, the work in ships, other than flag-ships, being quite within the scope of work of surgeons of a lower rank. (4) A real grievance seems to be that the flag allowance of second flag-ships, and in the flag-ships of the home ports, is not granted to the Medical Department, though all heads of other departments get it during the time the Admiral's flag is flying. We have no doubt that the Admiralty will remove what appears an injustice to the Medical Service of the Navy in their new warrant. (5) Allowance of time and pay for study-leave must be granted sooner or later, unless the medical men of the Navy are to fall behind in the matter of recent surgical and medical knowledge.

We are so proud of our Navy that we would grudgingly see the Medical Service of the Navy starved by neglect; and it behoves the civil members of the profession to write and speak for this branch of the profession, the members of which are wholly debarred from writing or speaking for themselves.

SCHOOL OF TROPICAL MEDICINE IN PORTUGAL.—It is proposed to establish a colonial hospital and a school of tropical diseases in Lisbon. There are to be three chairs created, namely, tropical pathology and clinical medicine, tropical hygiene and climatology, and tropical bacteriology and parantology.

Correspondence.

To the Editor of the JOURNAL OF TROPICAL MEDICINE.

DEAR SIR,—Your Editorial in the JOURNAL OF TROPICAL MEDICINE for December 2nd, 1901, is most opportune. Any one who knows the life on the west coast of Africa will admit that after even fifteen years' service there, there would be few candidates for pension.

Nothing can attract men into a service where one never knows but that within a few days one may be lying rotting in an almost forgotten grave.

Pecuniary advantages, leave of absence, hope of rapid promotion, transfer to a healthier climate, good pension coming quickly, better dwellings, less ordering about, may all cause a better class of man to come forward to fill vacancies.

At present, in places, it is hard to fill vacancies with any sort of man, and the Service is consequently not only greatly undermanned, but often men coming out are found to be such weeds that they are quickly sent home again, costing the Government a good deal of annoyance and expense. Often, especially in coastal towns, men are chosen to fill the stations because they can do administrative work. This to no small extent, hinders them in their own work, and a medical man, as a rule, does not relish sitting in court trying cases.

As most men come out here to make as much cash as possible in as short a time as possible, it is only fair that they should be well paid for these extra duties, but unfortunately, they merely get about enough extra pay as to pay their servants for the year.

Minor inconveniences could be mentioned by the score, and I am sure that this Service must be dealt with in a very liberal fashion before the Colonial Office can get any way suitable men to select from, and not men who are no use, nor men who only wish to make enough money to set up at home.

Yours, &c.,
VERITAS.

News and Notes.

BERI-BERI ON BOARD A BRITISH MAN-OF-WAR.—His Majesty's cruiser *Pomone*, 2,135 tons, under the command of Captain E. A. Simons, arrived in Bombay from Muscat, having left that port on January 16th. On arrival in Bombay there were 35 cases of beri-beri on board. Eleven, including Lieutenant G. B. Hadd, were landed and removed to the St. George's Hospital, and the rest were removed to the Military Hospital, Colaba, last evening. The *Pomone* was sent to the Government Dock the crew to the Sailor's Home, and the vessel was thoroughly disinfected.

"A MOSQUITO-CATCHING PLANT.—Such a plant is much needed in India generally and in Calcutta especially. The plant to which the Americans have applied this name is known scientifically as *Vincetoxicum acuminatum*. It is a beautiful hardy herbaceous perennial from Japan. It is in full flower at the very beginning of summer, at which time it is one of the showiest things in the garden, and con-

tinues in bloom for several weeks. It grows to a height of from one to two feet, a clump of slender stems enveloped in a cloud of star-shaped, creamy white flowers which are borne in short cymes. The plant belongs to the Milkweed family and its flowers secrete a viscid substance that attracts insects and especially mosquitoes. Once these insects alight upon the flowers and plunge their "beaks" into the tempting sweet, they are hopelessly entrapped. No matter how frantically they struggle they cannot release themselves."

In amplification of the above it may be stated that the plant belongs to the natural order *Asclepiadaceæ*, and would probably thrive in India, as many genera of this Order are natives of this country. Any way, an experiment might be tried with it.

H. ST. JOHN JACKSON.

Editor, *Indian Gardening and Planting*.
Times of India.

Current Literature.

Papers in the British Medical Journal of January 25, on Tropical Diseases.

III.—ON THE CAUSAL RELATIONSHIP BETWEEN "GROUND-ITCH," OR "PANI-GHAO," AND THE PRESENCE OF THE LARVÆ OF THE ANKYLOSTOMA DUODENALE IN THE SOIL. BY CHARLES A. BENTLEY, M.B., C.M. EDIN.

Medical Officer to the Empire of India and Ceylon Tea Company, Limited, Borjuli, Tezpur, Assam.

In the JOURNAL OF TROPICAL MEDICINE for March 1st, 1901, an article appeared under the heading "Water-Itch, or Sore Feet of Coolies," describing a disease with which all tea garden medical officers in Assam, Cachar, and Sylhet, are familiar. Apparently a similar affection is very prevalent on the sugar plantations of the West Indies, for in the Annual Report, by the Surgeon-General of Trinidad, for the year 1900, it is mentioned that "ground-itch is very prevalent during the rainy season on the sugar plantations, amongst the East Indian labourers." It is further remarked in this report, that although the disease is not a serious one, it is a matter of great importance to the planters, on account of the number of labourers it incapacitates from work.

The disease has been defined by Dr. Dalgetty (Sylhet), one of the writers referred to, as "A superficial vesicular dermatitis, which occurs epidemically among the coolies working on tea gardens during the wet months of the year, which solely attacks the feet, and which has a considerable resemblance to ordinary scabies." In place of this definition I substitute one which I think is perhaps more suited to the condition.

DEFINITIONS.

Ground-itch (synonyms : pani-ghao, water-itch, water-pox, water-sores, sore feet of coolies) is an affection of the skin confined entirely to the lower extremities, and probably always associated with the presence of the larvæ of the ankylostoma duodenale in the soil of the affected areas; endemic in Assam and the West

Indies, and possibly present in other parts of the tropics; characterised by its periodical epidemic appearance in the infected areas, coincident with the onset of the rainy season; with typical lesion consisting in a primary erythema, followed by a vesicular eruption which frequently becomes pustular, and in severe cases may result in obstinate ulceration, or even in gangrene.

PREVALENCE.

In Assam the disease is almost universally known by the name "pani-ghao" (literally, water-sore), a term which has been applied to it probably because it only occurs during the seasonal rains, when the earth is saturated with moisture. On tea gardens, where the disease has once appeared, it becomes a yearly recurring epidemic. Sporadic cases may occur as early as May, but the general outbreak does not usually appear until a month or six weeks later, and does not disappear until about the end of October, when the ground has become comparatively dry again. People of all classes are liable to contract the disease, although it is rare for a European to be attacked.

The disease may generally be said to attack the different individuals in a community with a frequency directly proportionate to their exposure to the source of infection. All observations point to the earth as being the infecting medium, and faecal contamination of the soil as being the most active agency in the propagation of the disease, which in over 90 per cent. of cases is contracted in the immediate neighbourhood of the coolie lines, as the collection of coolie houses is termed.

It is a well-known fact that the Indian coolie is not very particular in regard to the disposal of human refuse. He will not adopt any proper conservancy system, and latrines, if erected on tea gardens, are never used by the labour force. The coolie walks but a few yards from his dwelling in order to relieve himself. In this way the cultivated areas for some distance round the lines in most tea gardens is constantly being contaminated with faecal matter. The coolies themselves recognise that this accumulation of filth is the cause of the disease, and as far as possible they try to avoid exposure to infection by shirking work in the parts of the garden in close proximity to the lines. When obliged to walk over the infected soil they almost invariably protect their feet by wearing "kurrams," a kind of wooden sandal.

In this part of Assam the disease appears chiefly among the labourers who are engaged in hoeing; it affects the strong and healthy quite as frequently as the weakly and anæmic. Cases occurring among hoeing people almost always show the eruption on the instep and ankle. This is no doubt due to the nature of their work, which necessitates their remaining near the same spot for a considerable time, and ensures that their feet are in contact with the soil, while a constant shower of moist earth falls from the hoe upon their feet and ankles. Of the total cases, 75 per cent. occur among men. This is explained by the fact that during the time when the disease is prevalent, the strong men are engaged in hoeing, while the more weakly men, the women, and children are at work plucking.

Among pluckers, the disease is very much more frequently seen on weakly individuals and those whose caste customs forbid a high standard of personal cleanliness. Among this class of coolie the lesion is almost always confined to the underside of the foot or to the clefts between the toes, and can generally be traced to neglect to wear kurrums, or to want of sufficient care in cleansing the feet after return from work. Want of cleanliness may certainly be regarded as a strongly predisposing cause of infection, and this fact rather than any inherent susceptibility must be looked upon as the reason why anæmic and debilitated coolies so frequently suffer from the disease. The reason that so few robust plucking people are subject to the affection is no doubt to be sought in the almost universal adoption of kurrums by the better coolies, combined with careful washing of the feet immediately on leaving work. The weakly coolie is always dirty and negligent in matters of this kind. He often eats his food without washing his hands, and it is probably quite exceptional for him to wash his feet at night.

SYMPTOMS.

The first symptom is an intense itching and burning at the spot where afterwards the eruption appears. This is sometimes so distressing as to cause the sufferer to rub his feet on the ground or some other hard, rough surface in order to obtain relief. A faint papular eruption may be distinguished at this time, although sometimes a slight erythema is all that can be detected. This may occur within a few hours of infection, or may be deferred for twenty-four hours.

Later, usually about the second day, a distinctly vesicular eruption appears, which may be confined to the skin between the toes, or be found under the arch of the foot, or, as in a large number of cases, be chiefly seen in isolated patches over the dorsum of the foot, reaching sometimes several inches above the ankle. Apparently the affection is confined entirely to the lower extremities, no cases having been reported as occurring on any other part of the body, except as the result of experiment.

If early treatment is adopted the disease may be aborted, the vesicles drying up, and the patient becoming fit for work again in two or three days; more frequently the vesicles become pustules, or they burst, discharging a watery fluid, and then becoming open sores. If pustules have formed, unless they are speedily opened, the process may extend, the pus appearing to burrow under the surrounding healthy skin, and so forming large bullæ. In this way an area of several square inches may be denuded of skin.

In a large number of cases very severe inflammation occurs, associated with acute pain and great swelling, preventing the sufferer from walking; sometimes also extensive ulceration, or even sloughing, or gangrene takes place.

ETIOLOGY.

As previous observers have pointed out, this disease is one of great importance to the employer of coolie labour, for during the seasonal epidemic more than 5 per cent. of a labour force may be temporarily invalidated by its attacks.

Until recently nothing was known as to the cause of the affection, although various suggestions have

been made from time to time. Dr. Seheult (Trinidad) has stated that the condition is probably due to some chemical irritant present in the soil, either natural or due to manure used in cultivation. Dr. Dalgetty (South Sylhet), led astray by the superficial resemblance which the lesions of the disease bear to ordinary scabies, has described an acarus as the cause. His article in the JOURNAL OF TROPICAL MEDICINE was mainly devoted to an account of the anatomical characters and life-history of this mite, which certainly occurs as an accidental infection of neglected water-sores, and may almost constantly be found in the scab or crust round the edges of any untreated ulcer in this part of India.

I have been familiar with this disease in its clinical aspects for the past three years, have frequently met with it in tea-garden practice both in Cachar and Assam; but until this present season I have had no opportunities for investigating the nature of the condition.

I had been engaged in investigatory work for some two months before my attention was called to Dr. Dalgetty's article. After reading it, I felt greatly surprised that my observations appeared to be entirely at variance with those recorded by him. As a result, I repeated my experiments and observations, in order to satisfy myself as to their accuracy.

A few simple experiments served to show conclusively that the condition, although possessing a superficial resemblance to scabies, is in no sense an analogous disease. Dr. Dalgetty's methods of investigation are open to sources of grave error; thus he says: "If care be taken to evacuate the pustule completely, and to remove at the same time any crust that may happen to surround the spot, then in a certain percentage of cases the ova of an acarus may be seen." He adds that he has frequently found living acari in this way, which is hardly surprising, seeing that they may be found by the score on any dry dead animal matter which is exposed to the air. If, however, he does not find the mite in this way, "he lays the pus or crust aside for a day or two, covered up and moistened with sterilised water, but not covered with a cover-slip."

It naturally follows that very soon some mite in search of food finds its way to the specimen put aside for future inspection. I first found the acarus on the scab taken from an old water-sore, but on the same day I was fortunate to come across a colony of the same acari feasting upon the body of an *Anopheles* mosquito that I had put aside on a slide about a week before. Since then I have found the mite on all sorts of decaying animal matter, such as dry faecal matter, dead flies, &c.; and recently I bred about a hundred of them for experimental purposes upon the dead body of an ankylostoma.

It may here be remarked that the acarus described by Dr. Dalgetty seems to prefer dry to moist surroundings, and certainly it appears to breed very much more readily in the former.

In order to prove, however, whether or not the mite could produce the lesions of the disease, the following experiments were tried:—

Experiment No. 1.—A small quantity of soil was taken from a known infected area near to some coolie

lines, and after being moistened was applied to the arm, where it was kept in contact with the skin for a period of six hours, with the result that typical water-sores followed. The soil that had just been utilised, as well as the unused portion, was then carefully examined with the microscope, but no sign of any acari could be found.

This experiment showed that the cause of the disease must be sought for in the soil from areas near the coolie lines. This soil, as has already been mentioned, is contaminated with faecal matter in a very marked degree. Careful microscopic examination of samples of the soil from different areas was undertaken. The following list of living objects which were found present in these samples were noted down for future reference:—

(a) Rhabditiform larvæ of the ankylostoma duodenale (recognised by comparison with special cultures of larvæ) were present in great numbers.

(b) Minute leeches.

(c) Minute earthworms recognised by their setæ.

(d) Several species of rotiferæ.

(e) Several different species of infusoriæ, both amœboid and ciliated.

(f) The spores and hyphæ of several microscopic fungi.

(g) Bacteria, rod-shaped, curved, spiral (?) and micrococci.

While making these observations several further experiments were performed in order to show whether or not some chemical irritant in the soil might be regarded as the cause of the condition under consideration.

Experiment No. 2.—A small portion of the same earth that was obtained for previous experiments, and had been proved to contain the cause of the disease, was sterilised by being baked for a considerable time. Some of it was then moistened and applied to the arm as described previously. It failed, however, to produce any irritation, and thus completely negated the idea that a chemical irritant in the soil was the cause of the disease.

Microscopic examination of the soil in parts of the garden where the disease was never contracted showed that earthworms and minute leeches were present in greater number there than in the faecal infected places. Infusoria were to be found in pretty nearly as great numbers where decaying vegetable matter was present, and certainly could not be looked upon as a likely cause of the disease. Of the probable causes, some bacterium, some species of fungus, or the rhabdoid larvæ of the ankylostoma, had to be considered.

Meanwhile a large number of cases of water-sore had been carefully examined. In the earliest stage, when only a slight erythema showed where the vesicular eruption would shortly appear, blood films were prepared from the part affected. Nothing definite was derived from this procedure, although a few micrococci were seen once or twice. Scraping a patch of erythema showed on several occasions objects which at first were taken to be fibres of cotton or the shrivelled hyphæ of fungi, but when at last an intact and easily recognisable specimen of an ankylostoma larva was found, these objects were more carefully examined under a higher power, when they proved in a number of cases to be undoubtedly the empty

sheaths of similar larvæ. In the vesicular stage bacteria of certain kinds, lymph cells, a few blood cells, and once or twice a small amœboid organism were discovered. The pustular form of the disease showed staphylococci and streptococci, together with a few rod-shaped bacilli. Not having a microtome or other apparatus necessary for preparing sections, it was impossible to make this part of the investigation as complete as could have been wished.

Failing to find any definite cause for the disease by microscopic preparations for the lesions themselves, I once more turned my attention to the search for the cause in external Nature, and to this end the following experiments were undertaken:—

(a) Some ordinary soil was sterilised by heat, and after being moistened with sterilised water was infected with a small quantity of faecal matter, containing numerous ova of the ankylostoma duodenale.

(b) A similar preparation of soil was infected with a small quantity of faeces, which on examination were found to be free from ankylostomal infection.

These two preparations were incubated at the ordinary temperature of the air for about a week. At the end of this time sample "a" was swarming with larval ankylostoma and various forms of bacteria and fungi, and sample "b," but for the absence of the dochmii, presented a similar appearance. Of "a" and "b" respectively two small quantities were taken, the first portions of which were kept moist, while the second were gently dried at the temperature of the air for a period of eight hours. Previous experiments with cultivations of the larvæ of the ankylostoma had shown that gentle drying at ordinary temperatures for a period of six hours was sufficient to kill the larvæ. The four preparations were then applied (after re-moistening the two desiccated samples with sterilised water) to the wrists of the subjects of experiment, who retained them in a position for a period of about eight to nine hours. At the expiration of that time the bandages were removed. Fifteen hours after the first application of the earth, however, considerable erythema with a minute papular eruption appeared over the spot to which the earth containing embryo ankylostoma had been applied. Within twenty-four hours a distinctly vesicular eruption had developed, followed by pustules exactly resembling those found in the lesions of ground-itch. In the other cases a faint reddening of the skin was produced, which shortly afterwards entirely disappeared.

The small portions of soil which had been applied to the skin were then re-examined under the microscope. The sample which had been known to be swarming with the active embryos was found to contain no living larvæ, one or two dead specimens being all that could be detected. The sample that had contained dead embryos was seen to be full of their shrivelled bodies. Apparently, therefore, the living larvæ had entered the skin, and their entry had been followed by lesions similar in every particular to those found associated with the condition known as water-sore.

It now remains to review the fact which various observers have recorded with reference to the clinical nature of the disease, and see whether or not they appear to agree with the theory deduced from the

results of the experiments and microscopical investigation described above. The disease only occurs when the atmospheric temperature is fairly high and the earth is saturated with moisture; moreover it only occurs in areas in which, as has been already shown, there is constantly increasing faecal infection of the soil. Where the disease is prevalent ankylostomal infection is extremely common. Giles, Dobson, Rogers, and others, have shown that upwards of 75 per cent. of tea-garden coolies are the subjects of ankylostomal infection, and my own observations point to an even more general infection in certain districts. Thus recently among some three hundred coolies, in all conditions as regards health, whose stools I examined microscopically, only one individual showed freedom from ankylostomal infection. It may thus be readily imagined what an enormous number of larvæ of this parasite may be set free in the soil when the conditions are favourable to their development.

As I have previously shown, desiccation at comparatively low temperatures is sufficient to kill the larval ankylostoma; this is a simple explanation of the fact that water-sores are never seen during the dry season. At this time the ground is hard, and on tea gardens, owing to pruning and other processes of cultivation, is also denuded of the vegetation which affords protection from the rays of the sun during the rains.

The result is that the embryo ankylostoma—which hatches out from the ova within the first two or three days after they have been expelled from the host, in the faeces—perishes almost immediately.

The very rare occurrence of the disease among the rural population is explained by the different methods adopted in the bushes for the disposal of human filth. It appears probable that the acuteness of the inflammation attending an attack of ground-itch is largely governed by the nature of the organisms which accompany or follow the larval ankylostoma in its passage through the skin. That this entry through the epidermis can and does occur has been recently demonstrated by Dr. Looss and Professor Sandwith, of Cairo; but I believe that I am the first to identify this entry of the parasite as the cause of the distinct and well-known skin affection so prevalent in this and other parts of the world.

TREATMENT AND PROPHYLAXIS.

In the papular and early vesicular stage of the disease, the application of a strong solution of salicylic acid in collodion or methylated spirit will cause the eruption to dry up, and so cut short the attack of the disease to one or two days. If, however, pus has formed, the only treatment of any service is the opening up and disinfection of the pustules with pure carbolic acid, silver nitrate, or nitric acid, and the after-treatment of the sore as an ordinary ulcer. In cases attended with great swelling, inflammation, and a tendency towards the formation of sloughs, free skin incisions and the use of hot antiseptic foot-baths are indicated.

The adoption of a proper conservancy system would probably entirely prevent the occurrence of the disease, but to anyone familiar with the habits and prejudices of the Indian coolie this solution of the question appears beyond the bounds of possibility.

The wearing of shoes or some kind of protection to the feet is known to be an almost certain preventive of the disease, and the frequent application of tar to the feet of those engaged in working in infected areas is a procedure of proved value. It is probable that the application of sulphur, or some strong disinfecting agent, to the soil in the neighbourhood of the lines may prove to be of use in destroying the parasite and so preventing the disease.

I have to thank Dr. P. K. Mitra, my assistant, for considerable help in carrying out the various experiments described.

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Archives Russes de Pathologie, de Médec. Clinique et de Bacteriologie.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Brooklyn Medical Journal.
Caducée.
Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Esercito.
Hong Kong Telegraph.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
Journal of the American Medical Association.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Medical Record.
Medical Review.
Merck's Archives.
New York Medical Journal.
New York Post-Graduate.
Pacific Medical Journal.
Polyclinic.
Public Health.
Revista de Medicina Tropical.
Revista Medica de S. Paulo.
Sei-i-Kwai Medical Journal.
The Hospital.
The Northumberland and Durham Medical Journal.
Treatment.

Notices to Correspondents.

- 1.—Manuscripts sent in cannot be returned.
- 2.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.
- 3.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.
- 4.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.
- 5.—Correspondents should look for replies under the heading "Answers to Correspondents."

The Journal of Tropical Medicine.

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Original Communications.

NOTE ON THE SUPPOSED TRANSMISSION OF PLAGUE BY FLEAS AND OF RELAPSING FEVER BY BED-BUGS.

By GEORGE H. F. NUTTALL, M.A., M.D., Ph.D.

University Lecturer in Bacteriology and Preventive Medicine, Cambridge.

IN the paper by Professor Galli-Valerio, which appeared in the JOURNAL OF TROPICAL MEDICINE of February 1st, 1902, the hypothesis of Simond, that fleas may serve as agents for the dissemination of plague, is very properly exposed to criticism. I quite agree with Galli-Valerio in calling a halt. The evidence in favour of the "flea hypothesis" is worthless, and cannot withstand scientific criticism. I do not, on the other hand, wish to assert that fleas may not, under some conditions, transmit the infective agent. As Galli-Valerio points out, it is curious to see the ignorance displayed by medical writers as to the habits or existence even of different species of fleas. The main arguments brought forward by Galli-Valerio against Simond's hypothesis were already advanced by me in 1899, in the publication cited by him at the foot of page 34 of this Journal, leaving out, of course, reference to more recent publications. My experiments with plague, anthrax, mouse septicæmia, and chicken cholera were reported upon in the *Centralblatt für Bakteriologie* in 1897 and 1898 (vol. xxii., pp. 87-97; vol. xxiii., pp. 625-635), investigations being conducted with *Musca domestica*, *Cimex lectularius*, and *Typhlopsylla musculi*. I refer the reader to these publications for numerous facts upon which I cannot enter here. My experiments with flies clearly proved that these insects might be a source of danger in the dissemination of *B. pestis*. On the other hand, all attempts to infect mice and rats through the bites of freshly-infected fleas and bugs proved futile; on the whole, upwards of 250 infected

insects having been experimented with. The germs were digested in the alimentary canals of the insects, this taking place more or less rapidly.

Kolle has recently reported that he has obtained negative results from experiments conducted in a manner similar to mine. In other words, we have no trustworthy experimental evidence in support of Simond's hypothesis. Judging from experience in the manipulation of fleas and bugs in my experiments, there should be no especial difficulty in repeating them on a larger scale with plague-infected animals. It would, however, be a rather "ticklish" matter to conduct such experiments in a country free from plague. It seems to me that these experiments would perhaps make it unnecessary for Galli-Valerio to subject himself to an experiment. The first step should be to clearly determine if plague is transmitted from rat to rat, or mouse to mouse, by their own flea. I cannot agree with Kolle's statement that this question is mainly one of academic interest, the main point being that these rodents do disseminate plague. In all these questions a thorough knowledge is better than partial knowledge.

In Dr. Christy's paper (pp. 39-40, of this Journal) reference is made to a possible connection between bed-bugs and relapsing fever. As far as I know, Flügge (1891) was the first to suggest the possibility of vermin serving to spread this disease. Tictin (1897) was also imbued with this idea. He made experiments with bed-bugs, which he allowed to suck recurrent fever blood, and subsequently crushed, injecting the blood they contained into monkeys. When the blood was taken from the bug immediately after it had sucked, the monkeys inoculated with it acquired the disease; on the other hand, the result was negative with blood taken from bugs after an interval of forty-eight hours. Although the *Spirochæta* stained normally, they had ceased to be motile after forty-eight hours in the bug. Tictin did not push the observation further, but judging from my experiments with the various species of bacteria above

noted, the *Spirochæta* must have begun to degenerate. That they were subsequently digested is also highly probable. It seems to me, therefore, that no value can be attached to Christy's experiment on himself where he allowed himself to be bitten by bugs up to two weeks after they had infected themselves. I should suggest in any case a repetition of the experiment upon a suitable species of monkey, the bugs being transferred, as in my experiments above quoted, immediately after an interrupted meal, from a diseased to a healthy subject. We know nothing of the life history of the *Spirochæta* outside the body, but it is possible that being a motile organism it may, by its own movements, leave the mouth-parts or digestive tract of the bug for the body of the host whence the insect is sucking blood. Bugs are easy insects to manipulate. I have kept them without food or water for several months, and in this condition they will bite immediately when dropped into a test-tube which is inverted upon the skin of man or various animals (rabbits, rats, mice, guinea-pigs, chickens).

TWO NOTES ON MALARIAL FEVER IN CHINA.

By W. G. K. BARNES,
Staff-Surgeon, R.N.

(1) AFTER reading the article by Major Fitzgerald, R.A.M.C., in the *British Medical Journal* of July 15th, 1899, on the treatment of malarial fever by the inunction of creosote, I determined to give it a trial.

During the stay of H.M.S. "Undaunted" off the Wusung Forts for over four months in the summer of 1900, we had very numerous cases of benign tertian fever on board. In some of these I tried the inunction treatment, at first using 3ss of creosote to 3i of olive oil, but afterwards substituting lanolin for the oil. Pure beechwood creosote was used. The best case I had was one of the lieutenants. After using the creosote once, I explained to him that I wished to try and "cure" him without quinine. He was no stranger to the fever, and said he did not care what I did as long as I relieved him when he got the headache. Treatment by creosote only was persisted in for five days, but the daily attack continued, and I could detect no difference in the blood examination from day to day. Some of the men were also experimented on for from two days to a week, but in no case did there seem any signs of a "cure."

In all cases, after from ten to fifteen minutes, perspiration commenced and the headache and nausea (if present) disappeared absolutely. The temperature would drop from 104° to 99° or so in half an hour. Perspiration was excessive, so much so that in one case I had to change the man's mattress.

If the inunction was performed at the height of the fever there was no after-rise. If commenced with the premonitory chill (we hardly ever had a marked rigor), or before the temperature had attained its expected maximum, the temperature would still rise, but not to the height that might have been anticipated without treatment, and there would usually be no headache or nausea.

It seemed to me that there was little or no after-depression such as is seen after cold bathing or sponging.

To sum up: I concluded that the inunction is of no use as a "cure," but I should certainly use it on seeing for the first time a case of malarial fever with a temperature of 104° or over, preparatory to giving quinine. I also propose to try it, when opportunity arises, in the hyperpyrexia of pneumonia or other diseases.

(2) In 1897, while "hulked" in the "Tamar" at Hong Kong, we had a severe epidemic of malarial fever on board. In very many cases it was noted that when the tongue was protruded it was deflected most markedly to one side, usually the left. Surgeon Rock and myself both estimated that this symptom was present in about two-thirds of our cases.

MALARIAL FEVER AS MET WITH IN THE GREAT LAKE REGION OF CENTRAL AFRICA.

By ALBERT RUSKIN COOK, M.D., B.Sc.Lond., B.A.Camb.
Late Scholar of Trinity College, Cambridge.

(Continued from p. 58.)

The convincing proof was, however, yet to come. On March 19th a woman had been admitted suffering, it was assumed, from chronic malarial infection, parasites being readily seen in her blood. Only temporary improvement resulted, however, from quinine given by the mouth and hypodermically, or from Warburg's tincture. On April 8th the blood examination again detected parasites in spite of the quinine. On April 11th the patient succumbed to increasing asthenia. A careful *post mortem* showed unmistakable signs of typhoid fever, the lower part of the small intestine was studded with typical typhoid ulcers. Peyer's patches and solitary bodies were swollen and ulcerated. The long axis of the ulcers was longitudinal. The edges were overhanging and the bases sloughy, penetrating in one or two cases nearly to the serous coat of the gut. The ulcers were much more numerous and larger as the ileo-cæcal valve was approached; there were enlarged mesenteric glands; the spleen was enlarged, soft and pigmented; there were signs of recent bronchitis in the lungs; smears taken from the spleen, liver and mesenteric glands and stained with methylene blue, gentian violet, and methylene blue and eosin, showed malarial parasites to a moderate extent everywhere, sporulating forms being found in the spleen, and isolated rods here and there resembling either Eberth's bacillus or the B. coli.

I have given this case in some detail because the opinion is still strongly held that typhoid does not occur in Uganda, one doctor going so far as to assure me he could not believe it. Had he come to Mengo a careful examination of our preserved specimens would probably have convinced him. The fact is very important in the way of prophylaxis and avoidance of contagion, the stools needing careful disinfection, &c. The last three cases I have given are typical cases of a mixed infection—typho-malarial fever. Once armed

with this knowledge one could solve many a puzzling problem. During my absence from the hospital in 1898 the Sister left in charge admitted a case of apparent severe fever, who suddenly died with profuse hæmorrhage from the bowel. In the absence of any certain knowledge, I had remembered this case as possibly one of those fever cases associated with severe bowel hæmorrhage that are said to be met with now and then. I have little doubt now it was typhoid.

At the present time, July, 1901, I have under my care a far more typical case of what is undoubtedly typhoid.

The presence, then, of the parasite in the blood does not permit us to exclude typhoid, as a mixed infection may be present. In the latter case the temperature chart is often very irregular, being modified by the kind of malarial fever. It is hopeless looking for rose spots in a native, and in a European prickly heat, &c., often produces a factitious rash. Often the stools are typically pea-soupy, but constipation has been met with in several of our cases. Distension of the abdomen and tenderness over the ileo-cæcal region is very valuable, also the character of the headache, a dull headache being more characteristic of typhoid. I have observed marked tremors, too, in the latter case, and deafness not due to quinine. Bed-sores are very apt to occur if the native has been treated at home, and bronchitis is often met with. Lastly, the convalescence is far more protracted in enteric, the patient remaining weak for years. Phlebitis and pneumonia are not uncommon as sequelæ.

DYSENTERY.

It is merely important to notice that there is a form of malaria in which mucoid and blood-stained stools are passed which might easily be mistaken for simple dysentery, did not the presence of parasites in the blood show the necessity for quinine in addition to the usual astringent remedies.

Anomalous as it may seem, severe diarrhœa is frequently due to fever, and although these attacks often yield to astringents alone, yet it is of obvious advantage to attack the *fons et origo mali* and destroy the parasites.

The comatose or hyperpyrexial forms of fever have probably often been mistaken for sunstroke, and the delay thereby occasioned in having recourse to quinine may be fatal for the patient. Fortunately, a blood examination renders the diagnosis plain. One case occurs to me as a good example. A European residing in Busoga was known to have exposed himself to the sun, and was shortly afterwards found lying on his bed unconscious. As soon as possible a medical man was summoned, who, with commendable forethought, besides employing suitable remedies for sunstroke, which he was assured the attack was, took several films of blood which he gave me the opportunity of examining. On staining I found numerous typical parasites. The man made a good recovery under quinine, and careful enquiry elicited the fact that the first symptoms of illness preceded the exposure to the sun, and the latter was the effect, not the cause, of the illness.

PLAGUE.

Plague is endemic in Uganda, sporadic cases occurring from time to time, while occasionally an epidemic will break out and sweep away seventy or eighty people. The disease is usually limited, owing to the method in which the native villages are separated from one another. Here, again, the microscope is most useful. Only a short time ago a case of supposed plague was brought in with high temperature and enlarged femoral glands. The buboes were aspirated, and films made showed no bacillus pestis, while typical parasites were seen. Quinine rapidly ended the fever.

Small-pox, again, is endemic here, and now and again one is apt to be caught napping. One and a half years ago, when on a journey, a "boy" of my European companions complained of fever. I had no microscope with me. The usual remedies were given, but his temperature remained high, 103° to 105° F., and on the evening of the third day a few shotty papules were noted on his forehead. Four natives were vaccinated and he was carried by them on a litter to the capital, where he tardily recovered from a severe attack of confluent small-pox. Another case was more pardonable, for a mixed infection was present, the microspore showing the presence of parasites in her blood, and a typical small-pox rash being developed.

Perisplinitis, due to fever, is not infrequently diagnosed as plastic pleurisy of the left side, nor is this to be wondered at when it is remembered that the pain in the side, shortness of breath, friction rub, stabbing pain on cough, &c., are signs common to both.

Pneumonia, heart-failure, coma, meningitis, and bronchitis are only mentioned to emphasise that they are often present with fever, either concomitantly or as sequelæ.

Non-malarial fevers are a very puzzling class, about which very little is as yet known. Excluding the important class of abortive typhoid fever, we may say that their resistance to quinine, and the absence of the malarial parasite, distinguishes them from malaria.

PREDISPOSING CAUSES.

These have mostly been enumerated in the foregoing paragraphs. The most potent is a previous attack, then follow chills, over-fatigue, exposure to the sun, the presence of another illness, mental depression. The catamenial period is a specially dangerous one for women and the puerperium. A European lady, who assured me that she never had fever, had a high temperature the day after her confinement. I made a blood examination and found typical parasites. The temperature rapidly lowered under quinine.

AGE.

No age is exempt. Natives who live to a great age seldom seem to suffer, probably because they have, through multiple infection, developed a relative immunity. As regards the other extreme of life, I have seen cases of fever in children four, five, six and ten days old, confirmed by finding parasites in the blood.

PROPHYLAXIS AND IMMUNITY.

I have said nothing about the extra corporeal cycle of the fever parasite because I have had no opportunity for research in this direction. Every fact one comes across here verifies the brilliant induction drawn by Manson and verified by Ross, Bignami, Celli, Bastianami, &c. *Anopheles* is everywhere present in Uganda, the little black mosquito that we all dislike so much from its smarting bite, its low hum, and its persistent attentions being, I take it, *Anopheles funestus*. A phenomenon that puzzled me before the promulgation of the mosquito theory, namely, small, limited epidemics of fever, is by it easily explained. Four or five cases may come into the hospital from a particular house or group of houses within a few days of one another, all suffering from the same kind of fever. Doubtless there is a pool close to the house that breeds *Anopheles* larvæ. Coming to the practical point of prophylaxis, the bearing on it of the mosquito theory is evident. Speaking for Uganda, I may say that the prevention of mosquito bites by living in elaborately defended houses, with doors and windows covered with mosquito netting, is quite impracticable, for the simple reason that mosquitoes do not agree to bite only between the hours of 6 a.m. and 6 p.m., as they seem to do in Italy. It must be remembered that sunset and sunrise occur here, situated as we are on the equator, approximately at these hours all the year round. At Mengo, with the surrounding swamps well drained, and in well-constructed European houses, we suffer very little from mosquitoes. But when travelling—no infrequent occurrence of course—the swamps which intersect the country in every direction form a very happy hunting-ground for the mosquito. I have a vivid remembrance of a journey I recently made in North Uganda, the little pests buzzed in clouds round our heads and legs in the morning, and though they got fewer after 10 a.m. they bit occasionally, even at noon. It is, of course, essential to sleep inside a well-constructed mosquito net, and not to trust to a native servant to tuck it in. Pools of water likely to afford breeding-grounds for *Anopheles*, near the house, should be drained, and flower vases, saucers for plants, cisterns, &c., periodically emptied out.

The better prophylaxis then, for here at any rate, is the regular taking of a five-grain tablet or tabloid of quinine daily, in the morning with breakfast. If a person cannot be persuaded to do this (and the theory of Koch that blackwater fever is caused by over-doses of quinine has done harm that it will take many years to eradicate) he will probably agree to do so while journeying in a specially malarial region, or when feeling out of sorts.

The immunity secured by regular doses of quinine, though not perfect, is of a very high order.

Natives certainly have a relative immunity, though this is, of course, liable to break down, and as a class they suffer severely from fever. I find on examining the last thousand entries in the out-patient book that 110 (11 per cent.) were cases of fever. Out of 1,796 consecutive admissions to the hospital no less than 455 cases were of malarial fever (25.5 per cent.), with 53 deaths.

Their relative immunity is shown by the undoubted fact that one may often discover malarial parasites in small numbers in their blood without the clinical signs of fever. Whatever agency, whether phagocytic action of their leucocytes or anti-toxin products elaborated in the serum or spleen, or bone marrow, or elsewhere, may be at work limiting the growth of the parasites, it is liable to break down, and the unchecked division of the sporocytes soon leads to the outburst of fever. Presumably then, even these apparently healthy people may, through transtellation of their blood by an *Anopheles*, infect others.

The third series of reports of the Malarial Committee of the Royal Society shows most interesting facts in the study of immunity of the natives.

Unfortunately I have only seen a brief *résumé*, which has reached me too late for me to do much work on the same lines.

The Commissioners found that the proportion of native babies under two years of age infected by malaria was very large. In Uganda it is very difficult to ascertain exactly the age of the infant, as the parents never know, but I found parasites in the blood of every child under two which I tested. I, however, have so far only examined the blood of twenty-two infants (one was older, about three years). The majority of these presented the clinical symptoms of fever, but in nine cases the temperature taken in the arm-pit or groin ranged only between 96.4° and 98.5° F. Christopher and Daniels remark on this point: "Children may have the æstivo-autumnal parasites in their blood and yet appear in excellent health, and need not suffer from fever." This would be corroborated by the temperatures I have given above. They also lay stress on the high proportion of children with enlarged spleens. Taking 100 children under two years of age, I found that 40 had enlarged spleens. The children were selected quite independently of their having fever or not. Of 100 adults taken in the same way only 17 had enlarged spleens. Of those definitely ill of fever of all ages 44.8 per cent. suffered from enlargement of this organ. It is evident, therefore, that a very large proportion of children are infected with malaria, and they, as Koch has pointed out—an inference also drawn by Drs. Daniels and Christopher—must be largely the means of infecting the mosquitoes, and through them the adult. The practical conclusion the Commissioners draw is that there should be a separation of European and native quarters of at least 400 yards; an almost impossible condition out here, either for Government officials, traders, or missionaries.

SEASON OF THE YEAR AS REGARDS MALARIAL INFECTION.

The subjoined table gives the number of cases admitted during the several months of four successive years into the hospital:—

| | |
|-----------------|------------------|
| January .. 54 | July 37 |
| February .. 44 | August 38 |
| March .. 50) | September .. 20) |
| April 39) | October .. 32) |
| May 40) | November .. 14) |
| June 55 | December .. 30 |

Rainy season.
Rainy season.
Often rainy.

As a broad rule the dry season is the more danger-

ous of the two, but it must be remembered that rarely a month passes without some heavy showers, even during the so-called dry season; probably quite sufficient to leave pools of water, in which the larvæ of *Anopheles* may develop.

COMPLICATIONS.

Many of these have been described already, and it will be sufficient to merely enumerate them. Such are blackwater fever, diarrhœa, dysentery, severe vomiting, pneumonia, meningitis, coma, and bronchitis. The last and pneumonia are often due to chills caught during the sweating stage.

Malarial Amblyopia.—I have seen several cases of this in natives who have never had quinine, so that quinine amaurosis cannot have been a contributory cause. Unfortunately the patients I have seen came when already totally blind, and failed to improve under any treatment. I have been struck with the paucity of pathological appearances seen in such cases by the ophthalmoscope. Probably the lesions are largely microscopical. In the cases I have seen the blindness has been bilateral.

Neuritis.—Sciatica and facial paralysis, both usually unilateral, are not uncommon. The former being a common malady, it is a little difficult to be certain that it is due to malaria, but one case I saw in a European gentleman came on during the course of an attack of fever. He was most averse to taking quinine and suffered also from typical brow ague. When he overcame his objection to quinine he rapidly improved. Facial paralysis is probably common, but in natives it is difficult to get a reliable history. I have seen two Europeans who have suffered from it. In both cases it cleared up in about six weeks.

Neuralgia.—Not tic major but tic minor, or merely shooting intermittent pains, not very rarely develops in the subjects of chronic malarial infection. One European I saw was very bad with this, but recovered completely under quinine, arsenic and iron, and change of scene.

Peripheral Neuritis.—This is said to be very rare, but excluding minor degrees I have had one patient under my charge, a girl aged about 10, who exhibited the most classical form of the disease which directly followed on a severe attack of fever. There was severe pain on gently squeezing the muscles, absence of knee-jerks, paresis of the muscles of both legs and arms, simulating paralysis. She eventually made a good recovery.

Epilepsy.—This is very common among the natives. In one case under my care, a boy of about 4, a sharp attack of fever seemed to start epilepsy. Perhaps in this case it should be regarded as a sequel rather than a complication. On April 18th, 1899, he fell ill with fever, and was brought to the hospital on April 21st, with a high temperature and frequent attacks of convulsions. Parasites were detected in the blood. Under the influence of quinine the temperature steadily declined, but on April 27th he was having typical epileptic attacks. They quickly subsided under appropriate treatment and by May 8th had ceased.

Convulsions are of course common in children. Delirium is very common, especially with a high temperature.

Hyperpyrexia, *i.e.*, temperatures over 105° F. is also common, but responds very readily to treatment.

Gangrene.—This rare and interesting complication has been described in a paper by Professor Osler in the JOURNAL OF TROPICAL MEDICINE, December, 1900. We have had one case in the total number of cases of fever that have been under treatment during the last four years—some 5,000 in number. This was a man who came in with fever and developed gangrene of the arm, which progressed to a fatal termination.

Miscarriage.—This is a most common complication amongst the native women. The usual history is that they come to the hospital complaining of symptoms of miscarriage and suffering from fever. As they generally apply for treatment when the miscarriage is inevitable it is but rarely we can prevent it. Quinine acts very promptly in reducing the fever, and there does not appear to be any special liability to relapse during the puerperium in these cases. If the child has attained a viable age, *in utero*, it has a very poor chance of surviving, being handicapped even above the general run of premature children. A point to which I hope to turn my attention is whether maternal ague can be transmitted to the foetus by way of the placenta, as for example, small-pox or syphilis. *A priori*, I should imagine not, but the debility induced by the fever on the mother acts disastrously on the child. One would imagine that any malarial toxin produced by the manufacturing activity of the maternal parasites would circulate in the blood of the foetus.

Nephritis.—This is decidedly rare if the presence of actual renal casts be required as proof of the lesion. As stated above, I have found albumen in the urine of 74 per cent. of fever patients, but as a rule this entirely and rapidly clears up. The most I ever noticed was $\frac{1}{2}$ albumen. Here the fever was complicated by meningitis and the person died. One does not see what, for instance, is so common in scarlet fever, the plain and unmistakable onset of nephritis. Bright's disease is not common in Uganda, in 1,500 consecutive admissions into the hospital only 10, or 6 per cent., were diagnosed as cases of nephritis.

Phlebitis is not uncommon in chronic cases, especially in subjects weakened by insufficient food, &c.

SEQUELÆ.

The most common is severe anæmia. Repeated attacks lead to a very characteristic chronic malarial cachexia, called by the natives "Musana," which simply means daylight, and doubtless has reference to the lightening of the dark skin of the face.

A single sharp attack may much lower the number of corpuscles and their contained hæmoglobin. Thus in one European, a severe attack lowered his red blood corpuscles to 3,880,000 per c.mm. and hæmoglobin to 74 per cent. of normal (as measured by Gower's hæmocytometer and hæmoglobinometer). This was an uncomplicated attack of quotidian fever rapidly cured by quinine. It was not the first attack he had had in the country, however. After a course of iron for five weeks and a voyage on the Lake his blood values were 7,420,000 per c.mm. and 116 per cent. hæmoglobin. Children suffer even more. In a baby

about two years old, after fever the blood values were found to be 1,010,000 R.B.C. per c.mm. and only 17 per cent. hæmoglobin. Yet she eventually made a good recovery. In the typical case of severe anæmia the woman—for it is far commoner in women than in men—is pale, the face, instead of being black or deep chocolate colour, as the native complexion usually is, being more fawn coloured. The palpebral conjunctivæ are dead white, the ocular conjunctivæ being light lemon coloured. The tongue is pale, flabby, and often marked by the teeth. Loud venous hums in the neck and hæmic murmurs over the cardiac area are nearly always present. The spleen may be much enlarged, but sometimes is not. They are breathless and constipated, and their blood examination may show, in addition to great paucity of corpuscles and low hæmoglobin value, abundant poikilocytosis. In a word, they much resemble cases of pernicious anæmia. They frequently have attacks of fever. Pregnancy—and most of them seem to become pregnant—is very fatal; again and again I have seen their condition get worse and worse, they develop severe œdema of the feet and legs, and almost invariably miscarry about the fifth or sixth month and die.

One case bore a child at term, but the foetus was born dead and with skin peeling off. The next time she bore a living child, but she had been on large doses of iron for many months with occasional quinine administrations. One curious point about this class is that they are generally well nourished, not to say fat and well-looking.

Dementia and mania are well-marked sequelæ in Uganda. The prognosis is usually good, a large proportion recovering on quinine and nerve sedatives.

DYSMENORRHEA.

This condition is common amongst European ladies in the tropics. I have seen one case in which, absent at first, it seemed to develop after a long series of malarial attacks.

Blackwater fever seems never to develop, except in the subjects of malarial infection. It is often immediately preceded by many slight attacks of malarial fever.

PROGNOSIS.

In attempting to form an estimate of this most important question, it must be remembered that other factors besides purely medical ones crop up. An isolated European in the country may have been quite out of the reach of any trained nursing. An unconscious or delirious man, with only unskilled native servants to look after him, may have his life jeopardised, where proper attention and suitable food would have pulled him through. By the time the doctor has arrived, unchecked temperature or constant delirium may have rendered his work much harder.

Assuming common-sense precautions have been taken, in intermittent fever the prognosis is very good. Long-neglected cases may, however, suddenly take on pernicious symptoms. In mild remittent fever the prognosis is still very good. In the bilious remittent form it should be more guarded, and in severe remittent and the pernicious attacks every hour is of importance. In no other illness is the old Latin axiom more applicable—*Dum vita est spes est*.

Every medical man who has practised for any length of time in the tropics must have seen cases apparently in *articulo mortis* which have yet made a good recovery. Typho-malarial fever and meningeal symptoms render the prognosis very grave.

In blackwater fever the mortality is very high, but great care should be taken that anxiety felt by the physician should not be communicated to the patient.

TREATMENT.

Increasing experience merely deepens the conviction that every fair-minded man, sooner or later, arrives at, that in quinine we have the remedy for malarial fever. Different circumstances may modify the way in which it is given, or the dose for administration, but the patient whom quinine fails to cure is in a perilous position. From time to time new remedies are announced in this as in every other disease, but one by one are laid aside in favour of the old-fashioned remedy.

As regards, then, direct treatment by drugs, quinine in some form will be chosen. It is a very necessary precaution to see that a patient is not only taking but also absorbing the drug. In January of this year I was attending a lady suffering from severe tertian intermittent fever accompanied by much headache. She was taking 20 grains of quinine daily, but exhibited no signs of cinchonism, and the fever continued unchecked. On changing the sugar-coated pills she was taking for uncoated tablets, ringing in the ears quickly supervened and the temperature at once dropped.

In the case of young babies I usually order the quinine, from one to three grains, to be given in solution per rectum. In severe cases, if given per os, it had better be given in solution. A point that I have often noticed is that a dose of fifteen, or in some instances even ten, grains is sufficient to cause faintness and slight collapse, with damp, clammy skin, and most unpleasant subjective symptoms in some ladies. Under these conditions a stimulant should certainly be given along with the quinine. From time to time people will be met with who either cannot or will not take quinine. The former class is fortunately very small, much smaller than is generally supposed; with the latter, who are generally of a neurotic disposition, some stratagem must be used, provided a few straightforward arguments fail to set matters right. In the case of one gentleman who was suffering from most severe intermittent fever, the temperature rising with a sharp rigor of 105° F. every night, and who informed me that it was quite impossible for him to take quinine, saying it produced such an effect on his nerves, I told him I should have to give him some morphia injections. I need hardly say I dissolved very considerable doses of quinine in the morphia solution, and in two days his temperature dropped to and remained at normal. Subsequently I informed him what I had done, and he was quite convinced that he could take some quinine, at any rate without injuring himself. With some of these people combining potassium bromide with the quinine is advantageous.

In all cases presenting pernicious symptoms quinine should be administered hypodermically or intravenously. For hypodermic injection I dissolved

quinine—acid hydrochloride—the most soluble salt of quinine. Twenty minims of water readily dissolve 5 grains of salt. After trying various situations I prefer the interscapular region of the back. The skin should be sterilised and a platino-iridium needle used. A steel needle rusts inside in the tropics and it is then very difficult to sterilise it. Ten grains is a quite large enough dose, and it should be repeated every eight hours, if necessary.

Blood examinations should be made from time to time in serious cases to see if the parasites are duly disappearing.

I am trying methylene blue in various cases, but am not very much impressed by its value where quinine fails. Thus in a case now under treatment, a boy was admitted suffering from a tertian intermittent. He was placed on 15 grains of quinine daily, and while taking this his fever took on a remittent form. On July 2nd, seven days after treatment was commenced, the evening temperature was 103.6; the next night it was 104.8. July 4th, morning temperature 103.6. He was then placed on 2 grains of methylene blue thrice daily. Within ten minutes of taking the first dose he was found to be sweating profusely, and his temperature was found to be 97.8. That may have been mere coincidence, but his temperature never was more than half a degree above normal till July 11th, when it registered 99, and on July 12th (evening), it was 104.6, and July 13th, 105.2. He was then put back on quinine. If methylene blue be given to European patients it should be given in capsules to avoid staining the tongue, the digestion should be carefully watched, and above all they should be warned not to be alarmed if they pass bright green urine.

Arsenic, especially combined with strychnine and iron, is of great use in convalescence, and in chronic cachexia due to malarial infection. It is not nearly so useful in acute cases.

Where there is constipation the bowels should be kept open. Large enemata are dangerous in severe prostration, the patient sometimes showing signs of faintness, &c. Glycerine enemata are excellent, or a smart purge of calomel in a sthenic subject. Head-ache is best treated with phenacetin and antipyrin, but these should not be given in cases accompanied by a weak pulse. In such conditions I have seen very dangerous collapse take place, in spite of brandy having been given with the antipyretic. Hyperpyrexia is best met by cold sponging and cold affusions to the head. The latter is most grateful to the patient, and they remember the comfort it gives for years after. Sleeplessness is best met in an acute case by a hypodermic injection of morphia, with or without atropine. It seldom has to be repeated more than two or three times, as quinine will then have lowered the temperature. Of course, the patient should be on slop diet till the temperature has fallen. As regards dose and time of administration, much depends on the individual, but if the fever is severe a drop in the temperature should not be waited for, but quinine in full doses administered at once. In mild cases it will suffice to wait till the sweating stage. From 20 to 30 grains should be given in two or three doses per diem till the temperature drops, and then at least 10 grains daily for the next week. If no relapse

occurs 5 grains should be given daily for another fortnight.

Complications are, of course, met by appropriate remedies.

Hypodermic injections of $\frac{1}{2}$ grain strychnine are very valuable every four hours in heart-failure. Severe vomiting may be controlled by bismuth and soda, or in severe cases by one minim doses of tinct. iodi. in a teaspoonful of water every fifteen minutes till the vomiting stops. Champagne is often well tolerated. Delirium is usually much lessened by reducing the temperature. Acute splenitis is often soothed by hot belladonna and glycerine fomentations. With chronically enlarged spleens I generally use unguentum hydrargyri iodidi rubri, rubbed well over the tumour till the skin is sore, with quinine and, if necessary, arsenic internally. Anæmia requires free doses of iron, but the tongue should be kept clean, and in many cases of severe anæmia it seems but of small use. Here, again, arsenic may be added advantageously. In pregnant women large doses of quinine should be given with great caution, for it may act as an embolic. It is said that it is only the mineral salts of quinine that has this property. I tried tannate of quinine in one case. It only slowly reduced the temperature, however. In a first pregnancy, provided the temperature does not rise above 104°, miscarriage seldom seems to take place. In one case in which it took place the woman had had fever for three weeks, the temperature being about 104° several times. In blackwater fever the question has been most fiercely debated whether quinine should be given or not. Personally, I always start by giving full doses of quinine per rectum, if the stomach, as is generally the case, does not tolerate it. I also like to give a full dose of calomel and to promote diuresis and diaphoresis in every way. Should vomiting prove intractable I give nutrient enemata every four hours, and only give small quantities of aerated home-made lemonade to quench thirst, by the mouth. Very free exhibition of stimulants may be needed in this exhausting disease.

Typho-malarial fever demands the most unremitting nursing, night and day, if the case is to be carried to a successful issue.

In some desperate cases of fever change of situation has had the most favourable result, nor need the move be very far. I have known the change from a damp valley to the top of a hill, less than a mile away, apparently save life in two cases.

Invaliding home to Europe is nearly always necessary in the case of blackwater fever, and may be ordered in cases of chronic infection when other remedies have proved useless.

NOTHING can be more conclusive of the merits of an article than its successful sale and general use. Hartmann's Wood Wool Vaccination Pads are a case in point. Since January 1st, 1902, this enterprising firm has turned out and sold 15,000 dozens of vaccination pads per week, a fact that amply proves their utility and comfort in wear to those vaccinated. It may be added that these articles are recommended by the leading public vaccinators, and have been approved of by the Local Government Board.

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THE

Journal of Tropical Medicine

MARCH 1, 1902.

THE POST-GRADUATE COLLEGE AND POLYCLINIC.

SOME three years ago a small coterie of medical men, under the leadership of Mr. Jonathan Hutchinson, established a Post-Graduate College in London. The institution has for the most part taken the second of its baptismal names—the Polyclinic—which, whilst it conveys to some extent the kind of instruction provided, does not indicate that the institution is intended for post-graduate study. Be this as it may, however, the post-graduates in all parts of the Empire seem to have heard of the college and its work, for it has attracted medical men from all points of the compass. At the opening of the college many rumours of obstruction were in the air, and even now one occasionally hears waning evidence of antipathy. The college, however, is based upon the surest

foundation, for it is none other than that of imperative necessity. The need for such instruction as the college gives, owing to the rapid advance of science, is a settled fact, and the case for post-graduate instruction requires no advocate. The number of medical men who daily throng the clinics, lectures, laboratories, and practical classes held within the walls of the college, testify in a convincing manner to the demand for an institution of the kind; and it also shows a most noteworthy and healthy tone in the medical men of the present day that they avail themselves of such instruction.

The Polyclinic is purely a "medical man's" institution. It was founded, financed, and carried on by medical men; and it is the only public institution of the kind in the country that has such a basis.

Such being the case, it may be safely said that the reputation of the profession generally is bound up in this institution; and on its success or failure the good name of a great profession is staked. To allow the college to fail would cast a slur upon medical men, not only upon those who are actively concerned in carrying it on, but upon the profession throughout these realms and the empire generally.

Post-graduate teaching is a necessity; it must be conducted away from medical schools intended for students; and the London Polyclinic is the only serious attempt in Great Britain to meet these requirements. That the necessity for post-graduate teaching will continue for all time may be regarded as a positive axiom, and that an institution such as the London Polyclinic will be required in which to conduct the instruction is also a certainty; and it therefore remains to be seen whether the medical men of the present day are public-spirited enough, and sufficiently in earnest about their profession, to carry their college to a high platform of usefulness. That there are earnest men amongst us the attendance at the Polyclinic shows; it is no dilettante attendance either, for the medical men who attend there are not the idlers in medicine, but the most earnest and the hardest workers, and therefore the best, our profession has to show.

There is every sign that the Polyclinic is an institution that has "come to stay," and it behoves the profession generally to see that it is supported in every way possible. It is the duty of every practitioner in these islands to contribute to the support of the college, for with its success the reputation of the profession is intimately bound up. We have but one fault to find with this institution, namely, cheapness. For a guinea a year a medical man can attend daily clinics and many courses of lectures by men entitled to teach the subjects they deal with. Cheapness may seem a good fault, but the college has to be maintained, and to try to do this at the phenomenally small subscription of one guinea annually, considering what is given for it, is well nigh an absurdity. It must be noted that in addition to these clinics and lectures, the laboratories have to be maintained; the *Polyclinic Journal*, a monthly periodical, is given away to each member; reading rooms and a library have to be supported; and last, but not least, a museum of valuable clinical materials has to be provided with funds.

What attractiveness there may be in the small fee demanded tends to restrict the usefulness of the college, and runs the risk of hampering and crippling one of the most public-spirited institutions that the medical profession has ever taken upon itself to create.

To no class of medical men is the Polyclinic more useful than to those coming home from the tropics, and the large number of members recruited from thence shows how keenly the institution is appreciated by them.

A glance at the teachers and lectures in the college testifies to the quality of teaching given. The most distinguished men in the three kingdoms give their services, and the character of the instruction is therefore of the broadest and most catholic kind. The many cases now sent for consultation show how valuable the work of the Polyclinic has come to be, and how deep a hold the system in vogue has attained. The great want of the college, namely, a hospital, will one day be met, for it will be demanded by the great body of the profession. We therefore urge the

authorities of the Post-Graduate College and Polyclinic to throw aside their timidity and their fear of offending the more narrow-minded and the less public-spirited members of our profession, who condemn and are willing to sacrifice an institution, be it however useful, because it might interfere with the institutions with which they themselves are, in some way or other, connected.

The bulk of the medical profession have but little sentiment concerning institutions, however mediæval may be their origin and their so-called rights. The wants of medical men are proclaimed by the interest they take in the Polyclinic, and we are mistaken if at no distant date the Polyclinic becomes the most powerful medical institution in the land, and will be able to have all that it wants, be it a hospital, a Government grant, or anything that is necessary for the teaching of modern medicine.

Review.

TROPENHYGIENE MIT SPECIELLER BERÜCKSICHTIGUNG DER DEUTSCHEN KOLONIEN. Von Professor Dr. Friedrich Plehn. Kaiserl. Registrarsarzt, Z.D. (Tropical hygiene with special reference to German colonies, with medical advice to colonial officials, officers, missionaries, the leaders of expeditions, planters, and overseers. Twenty lectures given at the Seminary for Oriental Languages during the winter session, 1900-1901. By Professor Friederich Plehn. Illustrated. Pp. 283. Gustave Fischer, Jena. 1902.)

These lectures are intended as a guide to laymen who are so situated in the tropics that they cannot readily obtain the services of a physician. They will, however, be found of value to the physician when he first arrives in the tropics to take up practice. The lectures are explicit and simple, and should prove of inestimable value to those for whom they are intended.

The three first lectures deal with the tropical climates and their influence on Europeans. The four following lectures are taken up with malaria, its origin, prevention, and treatment.

Other subjects touched upon are blackwater fever, small-pox, plague, skin diseases, diseases of the stomach and intestines, animal parasites, snake and arrow poison, and diseases of the eye and ear.

The last six chapters give useful information concerning outfit for the tropics, tropical dwelling houses, daily life in the tropics, the hygiene of expeditions; and the last chapter is devoted to medicine chests and their uses.

Dr. Plehn has produced an excellent guide for the

tropics, and his book is sure to be popular with all laymen who have already gone, or intend to proceed, to the tropics, either for the purposes of travel or to take up permanent residence there.

"Assuring you of the sympathy and support of the other Medical Officers of this Colony,

"I am, &c.,

"A MEDICAL OFFICER,

"The Editor, "Colonial Medical Service.
"The Journal of Tropical Medicine."

News and Notes.

WE have pleasure in informing our readers that Lord Lister, Dr. Patrick Manson, C.M.G., and Professors Laveran and Virchow have been elected as Honorary Fellows of Société de Médecine de Gand, and that Major Ronald Ross, F.R.S., has been made Corresponding Fellow of the same Society.

Organisation of the Colonial Medical Service.

—[The Editors have taken upon themselves to withhold the name of the medical officer who sends this letter, although not instructed to do so by the writer. —ED., J.T.M.]

"SIR,—As Chief Medical Officer of one of His Majesty's Crown Colonies, I have read the article on the unification of the Medical Service in the JOURNAL OF TROPICAL MEDICINE of December 2nd with great interest.

"The scheme is one which should have the support of all Colonial Medical Officers, as the present system is unsatisfactory from many points of view, especially so from that of men working in the smaller and more distant colonies.

"According to the present system a man obtains an appointment in a certain colony, and is told on appointment that he has a chance of promotion in due course. Some time passes, and he finds that his advancement, with few exceptions, depends on vacancies occurring in his own colony. The colony is small; the vacancies are few. The consequence is, that after several years he finds himself still in the same place, and in receipt of the same salary, whilst junior men, quite new to the service, have been given appointments in other colonies to which his experience in the tropics would have rendered him eligible, and which if the Colonial Medical Service had been unified would have fallen to him.

"The pension and leave regulations also require serious consideration. They vary much in the different colonies, and Sect. 97 (of the C.O. Regulations, 1901) should be specially pointed out to each applicant for a medical appointment in the Colonial Service, as the clause referring to private practice considerably alters the amount, or may even do away with his chance of a pension altogether.

"I quite see that the difficulties in the way are great, and they may to some persons seem insuperable, but with united action a great deal may be done, and the opposition which this scheme is certain to meet may be finally overcome.

"It is, I presume, intended to form a Central Committee in London to try to carry out this scheme, concerted action amongst the widely-scattered Colonial Medical Officers being a practical impossibility.

Current Literature.

A PHASE IN THE HISTORY OF CHOLERA IN INDIA. BY ANDREW DUNCAN, M.D., B.S.(Lond.), F.R.C.S., M.R.C.P.

Physician to Seamen's Hospital Society; Joint Lecturer on Tropical Medicine, London School of Tropical Medicine; Physician to Westminster General Dispensary.

My first lessons on cholera were received at the hands of the late Sir George Johnson, at the time Professor of Medicine at King's College, London. The originality and lucidity of his theory of collapse, and the rationale of the treatment advocated by him, as well as the striking examples of the propagation of the disease by water narrated, had left an indelible impression on my mind. Judge, then, of the astonishment with which I listened when at Netley to the eloquent lecture on cholera by the late Professor Maclean, C.B., a lecture in which we were told of the views of the disease we should meet with in India. Professor Maclean rightly controverted this disastrous theory, and warned his hearers never to believe in them. This view certainly does not adorn a tale; nevertheless I venture to tell the tale.

India is pre-eminently the land of sacred pilgrimages. Let me narrate in particular the history of one of them. Every year to Hurdwar, the head of the great Ganges Canal, a pilgrimage takes place; but as every twelfth year is a specially sacred year, an especially large body of pilgrims wends its way thither. The ritual is the following: On the chief day of the pilgrimage, each individual by his religion is impelled to bathe in the canal, and a space divided off is set apart for the purpose; he not only has to bathe in it, but has to drink some of the sacred water before he leaves it; whilst, thirdly, some of the water is in numerous instances taken away in bottles to their homes by the pilgrims.

In the year 1867 such a twelfth-year festival took place. Just before the conclusion of the gathering, cholera broke out amongst them, and with the dispersal of the pilgrims a vast epidemic spread over India. The Sanitary Commissioner with the Government of India in 1867, in his subsequent report on the outbreak, thus writes: "First, in considering the causes of the epidemic, he decides it was not due to filth and bad sanitation, for the camp was kept far better than, and in marked contrast to, all preceding fairs. Secondly, it was not due to atmospheric phenomena. For there was nothing to show that there was any variation in temperature, or air pressure, or rainfall, different to those of preceding years. Thirdly, was it due to importation? There was

cholera in the Terai in 1867." The superintendent of the district writes: "The pilgrims took it to Hurdwar, and from Hurdwar they brought it back, and spread it on all sides. Numerous deaths occurred amongst the pilgrims proceeding to Hurdwar prior to the outbreak." In the Bhurtpore district, again, cholera was present on April 6th. The Maharajah of Bhurtpore visited Hurdwar with a large retinue just at the time when the disease began to be disseminated. The Sanitary Commissioner thus concludes in paragraph 40 that neither filth nor atmospheric causes were to blame. Hence, "if not general at the fair, it must have been brought there. This idea is quite consistent with facts; it is quite consistent with the etiology of other diseases, such as small-pox, regarding the propagation of which we have more precise information. Even had it been impossible to discover the probable source of the importation, the arguments would not have become invalid; but there is the direct testimony of the superintendent of the Terai, that pilgrims going to Hurdwar from his district had the disease among them and died on the way. It is sufficient to state that the disease, as it broke out at Hurdwar, appears to have been introduced by pilgrims from some infected district." Now the night before the great bathing day (April 12th) a heavy storm flooded the camp; the sewage arrangement favoured the washing of the sewage itself into the Ganges. On the day succeeding the storm the pilgrims bathed in the river, each pilgrim drinking of the water. The bathing-place was a space 650 feet long by 30 feet wide, shut off from the rest of the canal by rails. Into this narrow enclosure pilgrims from all parts of the encampment crowded, bathed, and drank of the water. The water was thus drunk foul and contaminated by whatever was washed from the bodies or clothes of the pilgrims. Cholera broke out in an epidemic form on the day after. I now quote again the remarks of the Sanitary Commissioner:—

"PAR. 300. *The effects of the dispersion of the pilgrims in spreading cholera.*—On this question the facts have been narrated with great care; every statement of any importance has been given, for the evidence has been considered not with the object of supporting any preconceived theory on the mode of propagation of the disease, but with the sole view of endeavouring to ascertain the truth. Did the pilgrims as they returned to their homes carry cholera with them, and thereby occasion the outbreak which subsequently followed in the various districts through which they traversed or to which they belonged?

"PAR. 301. On the first part of the question there can be no difference of opinion. That cholera went with the pilgrims from Hurdwar, and accompanied them to a greater or less distance in every direction, is a fact which admits of no dispute. Suffice it to say, that the pilgrims bore the disease with them to a distance varying from 50 to 300 miles in almost every point of the compass.

"PAR. 302. This fact in itself may be regarded as evidence of communicability of the disease. That the pilgrims imbibed the poison at Hurdwar in large quantities cannot be doubted, but it is not probable that the disease should remain latent so very long as to appear among some of them only when they

reached places so far distant as the Upper Provinces of the Punjab. Judging from all that is known of the disease, it appears much more probable that these and others who were seized weeks after they had left Hurdwar, were infected by pilgrims in whose company they had travelled, than that the germs of the disease had remained all that time undeveloped within their system.

"PARS. 303, 304. The results of the details regarding the advent of the cholera-stricken pilgrims, and the subsequent appearance of the disease amongst the general population of the districts who had been previously free from it altogether, may be thus summarised: Excepting Goorgaon, in which the history of the first case is doubtful, no cholera appeared in any of these fifty-one stations or districts until ample time had elapsed for the pilgrims to reappear, or for others to enter from infected places. There was no simultaneous outbreak of the disease over a large area; but the general evidence is not merely negative, for, excepting Goorgaon, there was no cholera in any of the fifty-one places named until the pilgrims actually had returned, and even in Goorgaon the epidemic prevalence dates from their return.

"PAR. 305. But even more remarkable is the evidence that in most cases the first cases in the district were pilgrims who had been to Hurdwar. In thirty-five out of the fifty-one districts the first persons attacked were pilgrims, and after they had been seized the disease appeared and spread amongst the residents.

"PAR. 306. In addition to the facts are the decided opinions of the numerous medical officers and civil officers by whom the facts were observed. Thirty-two medical officers, many of them men of great experience, who were indefatigable in carrying out the arrangements for the care of the devotees, and most careful in ascertaining the facts connected with the appearance of the disease within the limits of their own charges, are decidedly of opinion that the cholera was imported by pilgrims. . . . In no case has any positive evidence been advanced to show that such a cause was improbable, much less that it was impossible.

"PAR. 307. There are only two ways in which these facts can be satisfactorily disposed of. Either they must be set aside as untrustworthy, or they must be accepted as making out a very strong case in favour of the opinion that cholera is spread by human intercourse. It is quite impossible that the whole story of the returning pilgrims carrying cholera with them from Hurdwar to Rawal Pindi, with the dates of its appearance in the successive districts through which they passed, can have been invented. The facts cannot be set aside. It cannot be regarded as a mere coincidence that in thirty-five districts of Upper India, covering an area larger than that of Great Britain, the epidemic should have gradually appeared in one place after another, immediately after the return of a body of pilgrims stricken with the disease.

"PAR. 308. If not by the pilgrims, how was the disease spread? It could not have been carried by the wind in all directions at one and the same time, nor is it probable that the force of the wind should have exactly kept pace with the pilgrims. There are, no

doubt, difficulties to be explained under any theory which attempts to account for the fact.

PAR. 309. *Analogy of other diseases.*—But it is to be remarked that similar difficulties exist in regard to the spread of diseases, the communicative nature of which is undisputed. Can it be explained why small-pox prevails in some years and not in others? It is a singular fact that all epidemic diseases of which we have any accurate knowledge are communicable, and however fitful and inexplicable the cases may be, it may be fairly assumed that every new case is usually, if not always, the progeny of a parent of like kind, although the parentage often, and indeed generally, cannot be traced. The seed of a plant affords a not inapt analogy to what appears to be the most rational view of the germ of epidemic disease. In order to germinate and bear fruit, it must fall and be received in a suitable soil. It must be planted at the proper season, and enjoy the advantages of climate and circumstances which are best adapted to its growth. Similar conditions appear to be necessary for the propagation of an epidemic. With little doubt the germ of epidemic cholera appears to reside in the evacuation of a person suffering from the disease.

PAR. 311. But whatever theoretical difference of opinion may exist as to the propagation of cholera, the fact of the great epidemic of 1867, and its spread over Northern India, teach no doubtful lesson, and it is this—that human intercourse plays a very great part in the diffusion of the disease, and that returning pilgrims in particular are very dangerous arrivals."

This was the lucid summary of events given by the Sanitary Commissioner with the Government of India for 1867. The chief points to which he drew attention were the following:—

(1) The disease spread to every point of the compass. (2) The outbreak was not a simultaneous outbreak over a large area. (3) No cholera appeared in any place until the arrival thereof of infected pilgrims. (4) This fact could not be regarded as a mere coincidence. (5) In most places the first cases were among pilgrims, subsequently among the residents of the places attacked. (6) Out of fifty-one districts attacked, the medical officers were of opinion that it was due to importation in thirty-two cases, but that in none of the other cases was such a mode of origin improbable, much less impossible. (7) The conclusion arrived at, namely, that the return of the pilgrims is very dangerous, inasmuch as they play a very great part in the diffusion of the disease, and that the poison lies in their evacuations; that it is not due to bad sanitation or to atmospheric causes.

Such, then, is the history—so admirably portrayed by the Sanitary Commissioner with the Government of India for 1867—of one of the most striking examples of the propagation of cholera by human intercourse. As Professor Parkes truly declared, it was an example on a gigantic scale of cholera water poisoning.

Time went on, and with it a change of opinion occurred in the office of the Sanitary Commissioner with the Government of India. For in 1878 we find the officer who then held the appointment, in the orders of the Berar Sanitary Report, stating: "The chapter on cholera is too much occupied with the statements and opinions of civil surgeons on the

importation doctrine. Little or no practical good can be expected from such inquiries, and it is therefore of great importance that the time of the medical and other officers should not be spent on them. If it were demonstrated that cholera is spread by human intercourse, nothing could be done to prevent such intercourse." This was written, as stated above, in 1878.

In 1879 the great twelve-years' festival again came round. Again the vast assemblage of pilgrims took place at Hurdwar; again was cholera affecting them at Hurdwar; and again after the great day of the festival did "cholera burst out like a long-pent-up fire," and was carried away by the departing pilgrims to our North-Western Frontier, and thence to Afghanistan. But now, unfortunately, our troops were at war with Afghanistan. Camp by camp the disease invaded the three lines of advance.

In the Peshawar Column.—Thirty-eight cases occurred in May; 284 cases in June; 32 cases in July; 4 cases in October.

In the Kurran Valley Column.—Nine cases occurred in July; 12 cases in August; 1 case in September; 12 cases in October.

In the Candahar Column.—One case occurred in June; 52 cases in July; 41 cases in August.

Now, in reviewing these figures, one fact stands out pre-eminently, namely, that in the Khyber line there was nearly an eightfold increase of cases in June over those of the preceding month. In neither of the other two columns is there this remarkable increase of cases in any one month over another. Is there any reason for this? There is. Let me quote from Surgeon-Major Pringle:—

"From this intensely choleraised centre (Hurdwar), the outcome of the gathering of literally hundreds of thousands of pilgrims, with cholera proved to be in their midst, resulted what was known among the troops returning from the last Afghan war as 'the march of death.' Another proof of the penalty demanded for the violation of all sanitary laws and sanitation." The peace of Gundamack had been signed, and Government wished to withdraw troops from the Khyber line. But cholera was advancing up the pass. Most of the troops at this time were encamped on spurs above the plain, and were in good health. By remaining, it was held that they would become exposed to the "atmospheric wave," whereas by returning they would march away from it. By remaining, it was held they would be attacked in greater numbers. The withdrawal, therefore, took place in June, and the march of death resulted. "The choice was a choice of evils; and great as the loss was, there seemed every reason to believe that it would have been equally great, perhaps greater, had the troops remained where they were." But I venture to submit that the statistics of the disease in the other columns are not favourable to this opinion advanced by the Sanitary Commissioner with the Government of India. In the Kurram Valley no month stands out preponderating in cholera mortality, there being only nine cases in the British troops in the first month, and twelve cases in the second. In the Candahar column, counting the cases at the end of June with those of July, there was a decrease of twelve cases in the second month. In the Kurram

Valley, the troops moved out on to the surrounding spurs in most instances, and the result showed that, granting they were penetrated by an "atmospheric wave," the mortality was practically unaltered—nay, even in some regiments which lay more in the course of this wave in the plain, there was no increase of mortality at all.

We thus see that the views of the Sanitary Commissioner with the Government of India for 1878 and 1879 were diametrically opposed to those of the Sanitary Commissioner with the Government of India for 1867. By one, infection by human intercourse was held to be the cause of the epidemic; by the other, an atmospheric wave "was the *fons et origo mali*." Let us now, therefore, examine the arguments of the latter official; and such examination cannot be better carried out than in the spirit advocated by the Sanitary Commissioner for 1879. In par. 19 of his Sanitary Report, this official thus writes in 1879: "The problem to be solved requires patient investigation, and a calm judgment which shall not be swayed by any preconceived ideas or deterred by fear of adverse criticism. The cholera controversy has, unfortunately, excited much animosity and bitterness between those who differ in opinion. The doctrine of human intercourse is for the time the popular doctrine both with the medical profession and with the public, and neither facts nor arguments which are at variance with this view find much favour. All this is to be deeply regretted. If this doctrine is true, it can afford to stand the fullest criticism and the most thorough investigation." Animated by these inspiring sentiments, I propose to give in the following lines the fullest criticism and the most thorough investigation into the arguments and facts of the "climate and other cause theory."

The arguments for the "climate and other cause theory" may be thus enumerated:—

(1) The one-sided anecdote argument. (2) The X Y Z argument. (3) The railway and steamboat argument. (4) The hospital assistant argument. (5) The coincidence or stage-coach argument. (6) The logical method of reasoning argument. (7) The direction of the epidemic argument. (8) The hill station argument. (9) The *ipse dixit* argument. (10) The experience of India argument. (11) The non-spread by water argument.

(1) THE ONE-SIDED ANECDOTE ARGUMENT.—We have seen that in par. 19 of his Sanitary Report for 1879, the Sanitary Commissioner with the Government of India notes that the doctrine to which he is opposed can, if it be true, afford to stand the fullest criticism and the most thorough investigation. The following is an example of this fullest criticism. The careful investigations of authorities who uphold the theory of human intercourse are characterised as "one-sided anecdotes," and all facts adverse to the view are held to have been suppressed. Considering that the greatest investigators of our time are here implicated, such men, including that truly great man Parkes, Cornish, Burnell, De Renzy, Macnamara, Kenneth M'Leod, George Johnson, Simon, Neltén Radcliffe, Liebermeister, Koch, and a host of names of cosmopolitan celebrity, the accusation of "one-sided anecdotes" and of "suppression of facts" bears with

itself its own refutation. Again, was the Report of the Sanitary Commissioner with the Government of India for 1867 issued on a basis of "one-sided anecdotes"? It is indeed somewhat surprising that the large number of medical officers of 1867, who were then stated by the Sanitary Commissioner with the Government of India to have been "men of great experience, and most careful in ascertaining the facts connected with the appearance of the disease within the limits of their own charges," should have been succeeded by men apparently endowed with precisely opposite mental characteristics.

(2) THE X Y Z ARGUMENT.—"A hundred people are asked what is the cause of epidemic disease? Ninety-nine would probably reply that epidemics are due to contagion; that a sick person coming from some place or other brought with him the germs of disease, and that these germs found a fitting place for their development in the persons of other people. But now the hundredth individual, in the person of the Sanitary Commissioner with the Government of India for 1879, steps in, and says this is no explanation at all; for supposing a person Z is suffering from an epidemic, then there is little satisfaction in being told that it was imported from Y, and that the epidemic in Y was imported from X, for in time we shall get to A, and then where can A have got it from? Of this train of reasoning we can only say with gratitude that it would banish the whole body of infectious and contagious diseases from our midst. A poor relation to this argument is also to be found in the following: The case is cited of a person suffering from a disease, or coming from a place where such a disease is prevalent, and then becoming attacked by it. Shortly after his arrival in his new locality, one of those in his own house, or of those living in the neighbourhood, is attacked also. It is then usually judged—or at any rate suspected—that the first person has been the cause of the disease in others. Now this conclusion is at once totally rejected by the Sanitary Commissioner with the Government of India for 1879. Why? Because "there are others who have not suffered"! Following this line of argument, it will at once be seen how beneficial to mankind at large must be this elementary principle of Indian sanitation, for no infectious or contagious disease could possibly exist if this line of reasoning be true.

(3) THE RAILWAY AND STEAMBOAT ARGUMENT.—The Sanitary Commissioner with the Government of India for 1879 held that human intercourse has nothing to do with the diffusion of cholera, because railways have not facilitated the progression of the disease. Let me subject this statement to the "fullest criticism."

A. *Railways*.—(a) The Cholera Report of Dr. Rice on the outbreak of cholera in the Allahabad district in 1882 shows facts utterly opposed to this dictum.

(b) Deputy-Surgeon General A. B. De Renzy, C.B., the Sanitary Commissioner for the Punjab, showed how the frequency of cholera in the North-West Provinces increased since the introduction of railways. The dates of the epidemics show unmistakably how the latter increased in frequency after the introduction of railways. Thus the dates are as follows: 1805, 1813, 1827, 1845, 1856, 1861, 1862, 1865, 1867, 1869, 1872, 1875, 1876, 1879, 1881.

(c) The example narrated in support of this part of his argument by the Sanitary Commissioner himself apparently confirms his dictum, but "the most thorough investigation" does not do so. Thus—

| Station | Years | Strength | Admissions for Cholera | | | | | | |
|-----------|-------|----------|------------------------|------|------|-------|------|------|------|
| | | | Jan. to June | July | Aug. | Sept. | Oct. | Nov. | Dec. |
| Meair Mir | 1856 | 1576 | .. | .. | 435 | 65 | 1 | .. | .. |
| | 1861 | 1700 | .. | .. | 601 | 64 | .. | .. | .. |
| | 1869 | 974 | .. | .. | .. | .. | .. | .. | .. |
| | 1872 | 1358 | .. | 1 | 179 | 5 | .. | .. | .. |

The Sanitary Commissioner thus comments: "A glance at these cases will show that in 1869-1872, they occur no earlier than they did before in 1856-1861. At Meair Mir it is worthy of note that in the epidemic of 1861 the first case among the Europeans was on August 2nd. In 1872 it was on July 31st, a difference of two days." But in his comments on the above statement, Surgeon-General de Renzy, the Sanitary Commissioner at Punjab, draws attention to the remarkable fact that the details of the epidemic of 1867, *which began in May* and attained its maximum in July, and in which there were eighty-six cases with fifty-two deaths, is *omitted from consideration*, "although the influence of railway communication in diffusing it was most marked." Again, the same officer showed how no mention is made of the fact that in 1872 cholera appeared in May, and was most virulent in June in the lunatic asylums at Lahore, about five miles distant only from Meair Mir.

(d) Dr. Barry, in his Cholera Report for 1896, showed how increased facilities for steam communication by land and water have conduced to a rapidity in the diffusion of cholera hitherto unprecedented.

(e) In 1892 cholera appeared in Afghanistan. It became epidemic, and, marching slowly through Persia, it was at Teheran in 1892; once it touched Russian territory it spread along the railway with startling rapidity. At Askabad the epidemic came into contact with the Trans-Caspian Railway, spread eastward and westward along it, reached Baku, and thence found its way along the Trans-Caucasian Railway to Tiflis. Within a month of the recognition of the disease at a town on the Trans-Caspian Railway, it had penetrated into the heart of European Russia, having taken it in its transit from Central Asia *as many days as, before the creation of railways, it took months*. In its advance it clung in a remarkable way to the main lines of traffic, *i.e.*, along the railways and the Volga.

(f) In 1882, after the pilgrimage to Tirupati, Surgeon-General Burnell showed how cholera followed the dispersion of the pilgrims in all directions except towards the north; now there were no pilgrims going towards the north, as no railways led there, but his report on the subject evidently was not deemed worthy "of the fullest criticism, or the most thorough investigation into the arguments and facts,"

as Surgeon-General Burnell was invited to resubmit it. This, however, was not done by that officer.

B. *Steamboats*.—That ships do carry cholera is shown by evidence so strong that I must confess I do not see how it could be stronger. The evidence of all the epidemics that have occurred in England is irrefutable on this point. The same also with the outbreaks in America. To take one example. On October 10th, 1871, four days after the last case of cholera at Stettin, the ss. "Franklin" sailed thence for New York, with 486 'tween deck passengers, 55 cabin passengers, the ship's company, a cargo of merchandise, and 19 bales of rags packed in canvas. On the tenth day out cholera broke out. By November 6th, when the ship arrived in the quarantine harbour at Halifax, forty-two men had died of the disease. On November 6th and 7th, two men from Halifax went on board, and were employed in coaling and watering, and were "exposed for some hours to the poisoned air of the ship." Both were seized with cholera on the same day. From one of them the disease extended to his family, and then in widening circles through his village. No cholera had been present in Nova Scotia since the spring of 1866, when the ss. "England" had arrived with cases on board. This ship also brought the disease, the whole western hemisphere having been free for some years previously from cholera.

(4) THE HOSPITAL ASSISTANT ARGUMENT.—This venerable argument would seem apparently to be impervious to the effects of senility, and to ripen and mellow like wine. It is this: the hypothesis concerning human intercourse cannot be true, inasmuch as hospital attendants are never attacked with greater frequency than other classes of the community. Putting aside the fact that when occasion favours they are more attacked, the whole question is begged by this line of argument. For by it human intercourse is interpreted as contagion. But human intercourse may signify contagion in some instances, and may signify nothing of the kind in others. The same line of argument might be applied to enteric fever. Nurses do not as a rule catch enteric fever from enteric fever patients, and therefore it might be held that enteric fever is never spread by human intercourse. In order, however, finally to dismiss this argument, it is only necessary to state that cholera, like enteric fever, does not spread by contagion, as small-pox and the other exanthemata, but by infection as distinguished from contagion, by the sick infecting the healthy by means of the contagion of the sick entering the alimentary canal of the healthy with the food. The Sanitary Commissioner for 1877 in his report, par. 142, states as follows: "In order to prove that cases of cholera among attendants are due to contagion, it must be shown that attendants suffered in larger proportion than others." Accepting, then, this test of the truth or otherwise of the argument, let us now proceed to apply it.

(a) In the epidemic in Egypt in 1883, Surgeon-General Irvine in his report stated that the men of the Army Hospital Corps suffered out of all proportion to the men of the other branches of the service.

(b) Griesinger's statistics for Moscow for 1830 show the mortality amongst the hospital attendants was 30

to 40 per cent., whilst in the general population it was only 3 per cent.

(c) Laveran in Toulon in 1849 showed that out of 179 hospital attendants, 51 died of cholera, or 1 in 3, whilst in the garrison the mortality was only 1 in 15.

(d) In Oran, 1865, the mortality in the hospital staff was 8 per 1000; in the garrison only 1.66 per 1000.

(e) In 1865, at Toulon and Marseilles, the respective rates of mortality were as follows:—

| | | | |
|----------------------|-----|-----|-----------------|
| In the 22nd Regiment | ... | ... | 31.75 per 1000. |
| " 28th | " | ... | 29.8 " |
| " 38th | " | ... | 19.38 " |
| Hospital attendants | ... | ... | 38.60 " |

(5) **THE COINCIDENCE OR STAGE-COACH ARGUMENT.**—The theory that pilgrims from a cholera centre bring the disease is held to be as valid as a theory that in the old coaching days the coach brought the hoar-frost in the winter. Frost in the month of December, succeeding a date on which the coach from London had arrived, could be held to have been brought by coach on as valid grounds as that cholera attacking villages immediately after the arrival of cholera-infected pilgrims, could be held to have been imported by these pilgrims. Space forbids any consideration of this argument. Suffice it to say that the opinion of the Sanitary Commissioner for 1867 does not agree with that of the Sanitary Commissioner for 1879 as regards coincidence, as shown previously in the Report for 1867, par. 307.

(6) **THE LOGICAL METHOD OF REASONING ARGUMENT.**—We are told by the Sanitary Commissioner with the Government of India for 1879, that in the investigation of cholera our inferences ought not to be drawn from a number of *single* cases, but that rather the etiology of the disease should be adjudged from a consideration of the subject *generally* as a whole. But a whole is generally held to be made up of its parts; and if the single parts point to one conclusion, it may be safe to express the opinion that the whole points identically to the same opinion. Again, the Sanitary Commissioner claims that by virtue of his office he has enjoyed paramount opportunities for formulating his theory of cholera, that it is due "to climatic and other conditions affecting certain localities," inasmuch as he has been able to judge of India as a whole. He denies that the civil or military surgeon serving in cholera camps, in the plains, is capable of forming a correct judgment merely from local outbreaks. But the greatest master of geographical pathology, Prof. Hirsch, of Berlin, has enjoyed still greater facilities in this direction; and from a survey of cholera as it has appeared in *all* countries, he holds it communicable by human intercourse, and sums up thus: "That it is human intercourse which furnishes the media for this communication is proved on a large scale by the observations on the diffusion of the disease by pilgrimages and military campaigns, both in India and beyond it; whilst there are other conclusive proofs, furnished in innumerable instances from the smaller circles of diffusion."

(7) **THE DIRECTION OF THE EPIDEMIC ARGUMENT.**—As regards the epidemic from Hurdwar in 1879, it is argued by the Sanitary Commissioner for 1879 that

there is no connection between it and the dispersal of the pilgrims, inasmuch as very little cholera occurred in the Bengal Province. Unfortunately this is an argument that overreaches itself. Why did very little cholera occur in Bengal? For the very simple reason that very few pilgrims came from this part of India, and consequently very few pilgrims returned to it. Professor Simpson thinks that at most only one thousand out of nearly a million pilgrims came from Bengal.

(8) **THE HILL STATION ARGUMENT.**—With regard to the confutation of this argument, I cannot do better than quote the words of Surgeon-General A. C. De Renzy, C.B., a former Sanitary Commissioner of the Punjab. He thus writes: There are two surprising misstatements in par. 57 of the 9th Annual Report of the Sanitary Commissioner with the Government of India. The fact that the well-elevated hill station almost always escapes cholera, and the exceptions to this rule are inexplicable on the theory of human intercourse. After such a statement, it will surprise the reader to find that Murree, the most elevated of hill stations, has suffered terribly in three out of the eight epidemics which have visited the Punjab since the annexation. There are, in fact, few Punjab stations which have suffered more frequently or more severely. At p. 122 it is stated that the Murree depôt has suffered twice before from cholera, once in 1858, and again to a slight extent in 1867. It will be observed that in giving the previous cholera history of Murree, the statement is restricted to the history of the depôt, apparently for the purpose of ignoring the terrible sufferings of the native population of the sanatorium, in the midst of which to depôt stands." And Dr. De Renzy goes on to show that with regard to Simla a similar misstatement was made. Murree has, since these words were written, again suffered terribly from cholera. Dharmala, another hill station, also suffered from a very severe epidemic some years ago.

(To be continued.)

PLAGUE.

PREVALENCE OF THE DISEASE.

INDIA.—During the weeks ending January 25th and February 1st, the deaths from plague in India numbered 11,445 and 12,192 respectively. This shows a great advance in the death-rate from plague compared with the corresponding weeks in 1901, when the returns were 3,396 and 3,415.

The Punjab returns show the deaths from plague in this district to be 3,040 and 4,102 during the two weeks in question.

In the Bombay districts the figures are for the weeks ending January 25th and February 1st, 4,622 and 3,822.

There is a slight increase in plague in the cities of Bombay and Calcutta, but in neither are the returns so large as in the preceding year.

A plague riot is reported from Patiala, where the plague medical officer was threatened.

EGYPT.—During the week ending February 15th,

six fresh cases of plague occurred in Egypt and six deaths from the disease. During the week ending February 22nd there were seven cases and five deaths from plague.

The chief seat of the disease is at Tintah. Two cases have been reported from Kom-el-Nour, and one case in Alexandria. Since the outbreak on April 7th, 1901, there have been 304 cases of plague in Egypt, and 184 deaths from the disease.

MAURITIUS.—During the weeks ending February 13th and February 20th, there were fifteen and thirteen fresh cases of plague in Mauritius, and nine and eleven deaths from the disease, respectively.

CAPE OF GOOD HOPE.—During the week ending January 25th there were no fresh cases of plague nor any deaths from the disease notified in Cape Colony. Nine persons remained in hospital under treatment for plague on January 25th.

SHANGHAI.—The P. and O. ss. "Ballarat" was in quarantine on February 24th, at the Wu-Sung anchorage below Shanghai, with five cases of plague on board. Two of the patients succumbed to the disease.

AUSTRALIA.—Plague has reappeared in Sydney. Three deaths were reported during the week ending February 28.

POST-DYSENTERIC AILMENTS.—Haasler's observations were made at Tientsin, and his article is based on hundreds of *post-mortem* examinations conducted there. His vast experience leads him to conclude that death occurs in consequence of the destruction of the intestinal mucous membrane, the loss of power of the entire large intestine, or to peritonitis, hæmorrhage, and other complications. He is of opinion that injections are not advisable during the acute or ulcerating stage of the disease. Appendicitis is a danger that must not be lost sight of after recovery from dysentery, and contraction and stenosis of the intestine are frequent sequelæ. Circumscribed abscesses may remain and prove a source of relapse. Cardiac and renal symptoms seldom supervene as post-dysenteric symptoms, but pleurisy is common and the organs of respiration are frequently affected.—*Deutsche Med. Wochenschrift*, January 9th and 16th, 1902.

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Archives Russes de Pathologie, de Médec. Clinique et de Bacteriologie.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Brooklyn Medical Journal.
Caducée.
Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Esercito

Hong Kong Telegraph.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
Journal of the American Medical Association.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Medical Record.
Medical Review.
Merck's Archives.
New York Medical Journal.
New York Post-Graduate.
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The Journal of Tropical Medicine.

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Original Communications.

DURATION OF THE LATENCY OF MALARIA AFTER PRIMARY INFECTION AS PROVED BY TERTIAN OR QUARTAN PERIODICITY OR DEMONSTRATION OF THE PARASITE IN THE BLOOD.

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Definition of terms.—Latency of malaria should be considered as a state into which the parasite has passed so that it does not show its presence in the individual by such symptoms as are manifested when active.

This state of latency is due either to an inherent inability of the parasite to declare itself or to a condition of the individual infected, whose organism holds, as it were, the parasites, so that their power cannot be manifested. The microscope will often reveal the presence of such latent infections, but not always.

There may be, instead of a real latency, a *masked* form of malaria in which there are symptoms, and from operating causes they are atypical.

The investigation of the duration of the latency of malaria demands a careful study of the locality, as to its latitude, altitude, temperature, moisture, prevailing winds, water courses, contiguity to malarious districts, and all other conditions that oppose or encourage the growth and distribution of the malarial parasite.

Celli ("Malaria," page 157) says: "Malaria is, therefore, a local phenomenon which must be studied on the spot; and the data gathered in any particular territory cannot be generalised, or applied to all countries."

This leads me to say that I have accepted as proven

that the mosquito is the only means of infection. It seems to me the experiments of Drs. Sambon and Low in Ostia, and in other parts of Italy by Celli, Grassi, Fermi, the Red Cross Society of Italy, Bignami, and quite a number of others, prove this beyond question.

McFarland (*New York Med. Journal*, November 17th, 1900) says: "In order, therefore, that a person shall be infected with malaria, it is necessary that he shall be bitten by a particular kind of a mosquito and a definite length of time after it has become infected by the blood of a malarial patient."

Thayer (*Trans. Congress of Amer. Physicians and Surgeons*, 1900) says that the only proven way of malarial infection is by the mosquito.

Osler ("Practice of Medicine," 4th edition) seems to accept without reserve that the hæmocytozoa of malaria are transmitted to man only by the bite of the mosquito.

In order to prove that the parasite has been latent in any individual, it must be shown that the person has had no chance of reinfection. This is a difficult task to set oneself in this part of the world, where malarial fever is so widely prevalent. In older sections and countries where sanitation and better drainage have been secured, the question of the place where primary infection occurred should be more easily determined. There are, however, in this country localities that seem to be favoured by their location, conditions, and surroundings. Galveston, we may say, is one of that class.

I have depended upon my knowledge of the various localities in which the cases reported were exposed to the agents of infection, to determine the time at which the parasite entered the organism.

The manifestation of paroxysms occurring at the place suspected of being the one of primary infection, or soon after leaving such a locality, is taken as more conclusive evidence that the parasite entered the person while residing in or passing through that place.

The microscope has been relied upon entirely to ascertain whether these cases had a malarial infection or not. No case has been reported where I failed to demonstrate the presence of the parasite in the blood, no matter if a clear history of tertian or quartan periodicity was given.

Location.—Galveston is in lat. 29° 18' 17" N., long. 94° 47' 26" W., situated on the eastern end of an island twenty-eight and three-quarter miles in length, and not more than three miles in breadth at any point.

Altitude.—The island is not raised more than six feet above mean tide, thus making proper drainage a difficult matter. There are, on account of its slight elevation, quite a number of bays and small lakes in the western part of the city. The eastern and central part of the city has been filled, and is free of water except in the ditches following heavy rains. The island is bounded on the south by the Gulf of Mexico, on the north by Galveston Bay, on the east by the Gulf of Mexico and the channels from the bay to the gulf, and on the west by West Bay and channel.

The prevailing winds for the past eleven years have been from the south-east directly from the Gulf of Mexico. The summers are long and mostly warm. Winters are short and mild, usually having a short period, when the temperature goes to about 40° F., and, from October to March, occasionally it may reach freezing, and then again in a few days a spring temperature. During the short periods of cold weather the wind comes from north-west or north-east, but soon changes round to south-east again, followed by warm weather. So Galveston has alternately spring and winter weather from October to almost April.

The shortest distance of the island to the mainland at any point is opposite Virginia Point, where the railways cross the bay a distance of one and three-quarter miles. The next shortest distance is from the eastern end of the island to Bolivar Point, three and five-eighths miles.

Soil.—The soil of Galveston Island is of clean beach sand. Rain water quickly sinks below the surface of the ground. There seems to be a stratum of salt water just underlying the island, the depth to which depends upon the tide, varying from two to three feet below the surface. The city is, however, favourably located as far as proximity to other districts is concerned. If the malarial-bearing mosquitoes are not bred on the island, then they must be brought quite a distance across the bay into the city.

A few cases of malarial fever have undoubtedly originated here, but, from what can be learned, most of these came from the western part of the city. From three to six miles down the island are known foci of malarial fever. In 1900, of 481 cases admitted to the John Sealy Hospital, in which a blood examination was made, 153 of these cases were malaria. Of the 135 cases, in more than 90 per cent. their infection could be traced to some point outside of Galveston. A little less than 10 per cent. seemed to have become infected on the island. From January 1st, 1901, to November 1st, 1901, a more careful inquiry was made, and a closer record has been kept of the cases. Of the 421 cases admitted to the medical

wards of the John Sealy Hospital, where a careful blood examination was made, 53 of these proved to be malaria.

A diagnosis of malaria could not have been made in some of these cases other than by the microscope, as the symptoms of the coexisting diseases marked the presence of malaria. In order that the reader may get directly before him the result of this year's work, a table of these cases is presented, and by referring to an outline of the map of Texas, the point of infection in most of our cases may be located.

It will be noted that four cases, or 7½ per cent., received their infection in Galveston, and three of these, or 75 per cent. of those traceable to Galveston, received their infection in the western part of the city.

TERTIAN.

DOUBLE TERTIAN.

| No. of Cases | Place of Infection | No. of Cases | Place of Infection |
|--------------|--------------------------------|--------------|--------------------------------|
| 1 | Liberty, Texas. | 1 | Galveston, Texas. |
| 1 | Battle, Texas. | 1 | Trinity River. |
| 2 | Beaumont, Texas. | 2 | Unknown. Outside of Galveston. |
| 1 | Mexico. | | |
| 1 | Galveston (East). | | |
| 2 | Galveston (West). | | |
| 11 | Unknown. Outside of Galveston. | | |

ÆSTIVO-AUTUMNAL.

| No. of Cases | Place of Infection | No. of Cases | Place of Infection |
|--------------|--------------------------------|--------------|------------------------|
| 3 | Cleveland, Texas. | 1 | Clear Creek, Texas. |
| 1 | Olive, Texas. | 1 | Bryan, Texas. |
| 1 | Trinity River. | 1 | Kountze, Texas. |
| 1 | Houston, Texas. | 3 | Brazos River. |
| 1 | Belleville, Texas. | 10 | Beaumont, Texas. |
| 1 | Dickinson, Texas. | 1 | Tampico, Mexico. |
| 3 | Unknown. Outside of Galveston. | 1 | Little Rock, Arkansas. |
| | | 1 | Galveston, West. |

Again, to study the liability to reinfection in Galveston, I have made a careful search for the anopheles and its larvæ in suspected breeding places. While hundreds of the *Culex pungens*, *C. sollicitans*, and a number of *Stegomyia fasciata* have been found, only.

Six specimens of the *Anopheles crucians* have been seen by Drs. H. F. Sterzing, J. J. Terrell, W. L. Allison, and myself in and about the basement of the hospital. Other than these no other specimen of anopheles has been caught on the island. I visited at different times what appeared to be the most favourable breeding places of this genus of gnat, and systematically examined almost every pool of standing water in the city, but not one has been found.

I spent two days in Beaumont, Texas, a place from which, you will observe, about 23 per cent. of our total number of cases of 1901 came.

I not only found the adult *Anopheles maculipennis*, but found numerous breeding places of this mosquito in the city.

After carefully examining every ditch, pool, &c., in the neighbourhood of the hospital for breeding places, I came to the conclusion that those anopheles cap-

tered by us were brought into the city by freight cars which are hauled through malarial districts loaded with grain and cotton.

Many of the cars are brought within two or three squares of the hospital, and as all railways coming into Galveston pass through the western part of the city, this may in some degree account for the greater number of cases of malaria that originate in that part of the place.

I have recently observed mosquitoes enter a passenger car and travel with us for two hundred miles. The transportation of mosquitoes by baggage is referred to by Harvey in *Med. Record*, June 22nd, 1901.

I found a breeding place and quite a number of the *Psorophora ciliata* in the west end. This genus of gnat was also found in large numbers near the Beaumont oil-field, a very malarial district, but in whose immediate vicinity I found no anopheles.

I succeeded in infecting four *psorophora* from a case of æstivo-autumnal fever, and two days later found the parasites, hence I merely suggest that this mosquito may also carry the infection and be responsible for some of our cases. I present twenty-three cases, all of which, except four, are from observations made in the service of Professor J. W. McLaughlin, University of Texas Medical Department, to whom I make acknowledgment for his kindness in allowing me to use them in the preparation of this paper.

Case XII. was kindly furnished me by Dr. J. H. Ruhl, of Galveston. The other cases, I., VIII. and IX., were from my own practice.

Two of the twenty-three, or 47 per cent., were tertian at one time, 5 per cent. of these becoming later on double-tertian. Twelve of these cases were æstivo-autumnal, showing either quotidian or tertian parasites.

Tendency of Malarial Fever to Relapse.—Frequent reference is made, by almost all writers who have observed many cases of malaria, to the disposition or tendency for them to relapse. These observations were made by the earliest writers; while they were probably closer observers of many phenomena than we, would we not now, knowing the pathogenic agent, accept the clinical evidence that is frequently given of latency and relapses. The microscope must be used to make our diagnosis conclusive.

Fagg ("Principles and Practice of Medicine," 1st edition, vol. i., p. 242) speaks of relapses in the following words: "There is a strong tendency to relapse, not only under a fresh exposure to the ague poison, but even independently of it."

Marchiafava and Bignami ("Twentieth Century Practice") say that the same conditions that favoured the primary infection favour a return of the fever.

There are, in fact, but few cases that are allowed to run their course untreated for any length of time, or are poorly treated, but that relapse, whether remaining in a malarial district or removing to some other place. Cases I., VIII., X., and XI. are good examples. I have often seen cases of simple tertian fever where the symptoms could be completely controlled by the administration of doses not exceeding five grains of quinine per day for three days, but would soon recur. The period of recurrence is usually in some multiple of the tertian paroxysm. *Æstivo-autumnal* types are

more apt to recur than the tertian or quartan, being much more resistant to treatment.

Circumstances Modifying or Determining the Period of Latency.—A sudden change of weather, a chilling of the body, overheating and overexertion are known to cause a relapse. I have known workmen who had previously had malaria to have a recurrence every time they were put to work on the roof of a building exposed to the hot sun.

Hertz (Ziensen, "Cyclopædia of Medicine," vol. ii.) calls attention to the fact that paroxysms occur between 12 o'clock midnight and 12 o'clock noon, when the organism has gone longest without food. Persons who have been compelled to live upon scanty rations, as soldiers on the march, and others who are unable to get a sufficient amount of food, are subject to relapses following the lessened food supply. A latent infection may become an active one at almost any time by extraordinary strain being thrown upon the organism.

Manson ("Tropical Medicine," p. 7) has this to say: "This much, however, we do know, namely, that physiological strain or vital depression in the host tends to bring about conditions which break up, and that quinine and vital vigour tend to bring about conditions which favour latency."

Case VIII. had a relapse at the time of his mid-winter examinations. Medicines were taken and paroxysms stopped, but they again returned at the time of his preparation for final examinations, April 1st and 2nd. Two cases, No. IX. and No. XII., are properly "post-operative" malaria.

Welch ("American System of Practical Medicine," vol. i.), while admitting that reduced conditions of the patient from operation might and do favour recurrences, yet in the first seven years since the opening of John Hopkins Hospital not a single case of post-operative malaria has occurred.

Writers on surgery have called attention to relapses of this nature, but the literature I have been able to collect has presented no cases where a blood examination was made. I agree with Welch, Thayer and others that the symptoms of malaria following an operation with the temperature yielding to quinine do not justify a diagnosis of post-operative malaria unless the parasites are found in the blood.

Clinical Manifestations of Relapse Uncertain.—Many times the symptoms of a relapse are entirely masked by the presence of some other disease, or the symptoms are so atypical that one is misled.

Case I. was taken with nausea and vomiting while at school. I did not suspect malaria, but examined his blood only as a routine measure of diagnosis.

During the year 1900 we had in John Sealy Hospital the following cases where the symptoms of malaria were entirely masked: amoebic dysentery, lumbago, mitral regurgitation, and enterocolitis. During the year 1901 I have seen the following: neuritis, asthma, and typhoid fever, saying nothing of a number of cases that have had symptoms of other diseases associated with those of malaria.

Bell and Stewart (*JOURNAL OF TROPICAL MEDICINE*, September 2nd, 1901) report nine cases where malaria seemed to relapse from injuries received.

In Case IX. the patient had suffered more or less

with pain, which I thought was due to other conditions. These symptoms were relieved entirely by the first operation, and the effect of the second operation was to cause a relapse of malaria. The time between the last chills and fever, January 24th, to the manifestation of symptoms, June 30th, was 96 days. I searched the house for anopheles repeatedly, but none were found.

Case XII., who last had a chill in the latter part of July and was treated, showed no evidence of malaria, but the parasite was detected by a blood examination which was made eight days after operation on account of a slight rise in the temperature.

Trousseau observes that the paludal diathesis does not always manifest itself by fevers, but may announce its presence by organic changes of various kinds in the body.

The Relation of Acclimatisation and Immunity to Latency.—The Colorado, Brazos, and Trinity river-basins are known as very malarious districts. I have many times observed that persons after a long residence in these places seem to acquire the power of resisting the action of the parasites while residing in those districts. You may take one of these persons who has had repeated attacks of malarial fever, and who has lived in a malarial atmosphere, and send him to an equally malarious district in company with a person who has lived further north in a non-malarial country; the latter will be struck down, while the former escapes. This generally accepted idea has been well illustrated in Beaumont, which is located on the Neches River. In the early part of 1901 oil was discovered about six miles from the above-named city. The place soon became famous throughout the civilised world as the most wonderful oil-producing region known. Thousands of people flocked there from every section of the country. Almost every new-comer was struck down within a few weeks with malarial fever in some form. Many who have gone there to live say that they do very well after getting acclimatised. While the old citizens of the place have attacks of fever now and then, the percentage is small compared to the new-comers from non-malarial districts.

One of the Texas State prisons is located at Huntsville, where malaria occurs now and then in the town; but when the prisoners are sent out to the State farms on the Brazos River, where malaria is much more intense, they are stricken down with the disease almost immediately. After residence there of several years they seem to become more resistant to the disease. Manson (*"Tropical Diseases"*) attributes this acclimatisation more to education and experience than to any peculiar change that might take place in the organism rendering one less liable to attack. What he says is in most cases true, but not altogether, for prison life is much the same, whether a convict works on a sugar plantation in the malarial basin of the Brazos, or within the confines of a healthful district.

Manson (*The Practitioner*, March, 1901) holds that repeated infections produce more or less immunity. This, however, does not seem to be applicable to all cases living in malarial districts. In some persons

it seems that each attack only makes them the more susceptible to reinfection or relapses. If repeated infections produce immunity, why are many cases able to live in an intensely malarial place, yet cannot even visit a non-malarial district without having a relapse.

Dr. J. W. McLaughlin, Professor of Medicine in the University of Texas, gives us a striking example of this kind. Mr. Rab, who lives near La Grange, Texas, where almost everyone seemed to be suffering from malarial cachexia from repeated infections of malaria, was not able to visit his relatives at Austin on account of having a relapse every time he did so. Austin is known to be almost free of malaria.

Case V. is a good example of the effect of going into a non-malarial district from one where repeated infections had immunised him against relapses or new infections. After an absence of 40 weeks from his malarial district a relapse came. From the great size of the spleen I cannot help feeling that the parasites had been latent during a year and a half, and that some influence which we do not understand was operating while in Dallas, but when he reached here this influence ceased to act, hence the relapse.

Among the students who attend the Medical Department of the University of Texas located in Galveston, we have frequent relapses just after entering school. There is said to be a racial immunity. The negro race is known to be much more resistant to infection and relapses than the white people. I am sure from personal observations that they can live along the river basins in excessively malarious districts with impunity, while white people do not dare to go there. It would be an interesting study to make blood examinations of all persons residing in a malarial locality, to determine the matter of racial immunity or latency.

Cases II., X., XIII., XIX., XX., XXI., XXII. and XXIII. illustrate this susceptibility of persons going from a non-malarial district to one that is malarial.

How is Latency Explained?—Thayer (*"American System of Practical Medicine,"* p. 128) says that relapses are clearly proven to be due to the fact that all the parasites have not been destroyed by treatment, or by what Manson calls the "protective power of the human body."

But what is it that enables these parasites to remain inactive, and yet alive, ready at any moment at no fixed period, so far as we know, to begin again their multiplication? Why do the phagocytes, which seem ever to be on the alert to destroy all foreign micro-organisms in the circulation, allow the few malarial parasites to calmly rest in the bone marrow, spleen, or lymph glands? Have these phagocytes so much else to do as protective guards of the system, that they do not even attempt the destruction of the latent parasites? If phagocytes are responsible for many spontaneous cures, why is it they do not destroy the sporozoites when they first enter the blood from a suctorial insect? These are difficult questions to answer. Many theories have been advanced attempting to show why we sometimes have these long periods of incubation and latency while in other cases there are only a few days of incubation and no recurrence. Celli gives an instance of the incubation period of quartan infection lasting 47 days.

I have watched repeatedly for sporulation or flagellation of parasites after being taken into the body of a phagocyte, as held by Golgi, but in all my observations the apparent destruction of the included parasite followed. Any chance of further development was destroyed, or at least checked.

Bignami's theory of some resistant spore-form being responsible for latency is to me the most reasonable. A complete discussion of the intricate processes here involved would require more time and space than can be given here, but I cannot refrain from adding a few words in an attempt to give a solution.

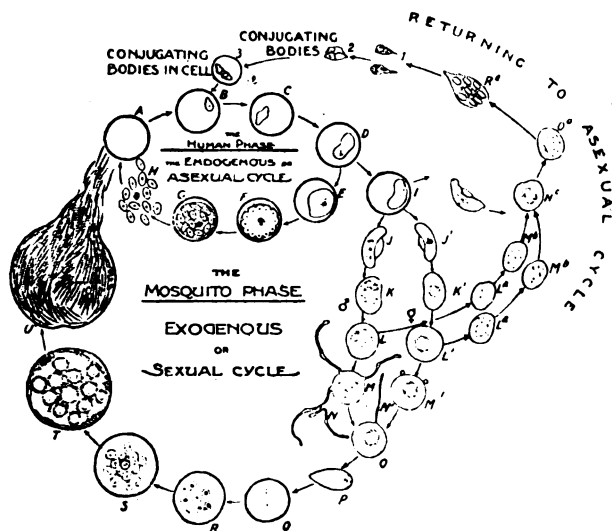


FIG. 1.—Modified schema showing what may possibly take place if bodies are not given an opportunity to flagellate and to fertilise the female bodies. Schema showing the human and mosquito cycles of the malaria parasite. Modified from Blanchard's diagram illustrating life cycle of *Coccidium Schubergi*. (Manson, *Practitioner*, March, 1901.) A, Normal red cell; B, C, D, E, red cells containing amœbulæ or myxopods; F, G, H, sporocytes; J, K, L, M, microgametocytes or male gametes; J', K', L', M', O, macrogametocytes or female gametes; N, N', microgametes; P, travelling vermicle; Q, young zygote; R, S, zygomeres; T, blastophore; U, mature zygote; V, bodies returning to asexual cycle; M^b—R^c, suggested phases; 1—3, small bodies that may go to form conjugating bodies.

To get clearly before the reader my views in the shortest space I shall take the liberty of using Manson's "Modified Schema" (*The Practitioner*, March, 1901) from Blanchard's Diagram. We shall begin with the amœbulæ as seen in the red blood-cells following infection with sporozoites by the mosquito. This endogenous asexual cycle goes on until we have generated a sufficient number of parasites to produce a paroxysm, or in some other way to manifest their presence by symptoms. This phenomenon presents no difficulties. A time comes, variously estimated by different authorities, when the parasite for some reason turns aside from the asexual cycle of segmentation to prepare itself for development outside of the organism. The tertian and quartan forms become "active" and "passive," or make flagellating and final forms. The æstivo-autumnal bodies, instead of going directly into their spherical flagellating and non-flagellating forms, pass into a crescent form which soon assumes an ovoid or spherical shape. Similar

spheres are seen in the tertian and quartan types of parasites. Could it be ascertained just what causes the switching off from asexual to those of sexual ones great strides will have been made towards the solution of the problem of the duration of latency.

Marchiafava, Bignami, Thayer, and others teach that these crescent bodies can be recognised when they are in a red blood-cell. If this be true, then each sporozoite must have been either a form going on to segmentation or a crescent body going on to the formation of male and female elements. If the kind of body that a sporozoite is to form is not predetermined, then at some stage in its progress some influence must act upon the amœbulæ to decide its final destiny. This could hardly be true, because, as far as observation goes, these crescent bodies do not begin to appear in the blood for several days after infection. Thayer gives this period as one week or more.

I have many times had recent æstivo-autumnal infections under observation in which neither the crescents nor the young crescent forms could be found, but after several days' treatment these would appear along with the forms which segment. Cases XII., XVI., XVIII., XIX. and XXIII. are good examples.

In my notes and drawings of studies carried on during the past year, I find that ovoids have often been observed to change to crescents; then, without any apparent reason for doing so, would reassume the ovoid shape. In one specimen this change back and forth from an ovoid to a crescent and a crescent to an ovoid was seen several times.

I believe most authorities hold that crescents never sporulate. There is much evidence in favour of this idea of sporulation of spherical forms that are crescent derived. Still, such authorities as Antolisei, Angelini, Grassi, Polletti, and others have contended that such is the case.

I cannot help believing strongly in Golgi's hypothesis (Ziegler's "Beiträge," 1890, VII., 647) that the crescents represent a form of parasite which undergoes a slow development or "process of internal differentiation, and finally sporulates." Marchiafava and Bignami, however, take the position that these observations of segmentation were processes of disaggregation or vacuolisation due to degeneration of the parasite.

We shall return to our diagram. Now if from some operating cause there occurs a differentiation of asexual segmenting bodies into active sexual bodies, might not this condition be reversed, and bodies that had gone far towards a sexual differentiation undergo some change reverting to an asexual form?

The diagram represents well what occurs in case the blood is withdrawn from the body by the mosquito, or as seen by the microscope. But in case these bodies remain in the circulation when they reach the crescent, ovoid or spherical stage, what happens?

Flagellation and fertilisation do not take place within the body of man so far as is known, so as to preserve the species. Then would not all the parasites soon become crescents and ovoids in a short time, thus limiting the growth of themselves, unless a mosquito happened along to take up and carry

forward its development? Nature does not take chances of that kind if she can help it. The presence of these small bodies, marked A in figs. 1 and 2, which apparently came from these crescent-derived spheres, that seemed to sporulate, has caused me to go back to the lower forms of vegetable life for an explanation.

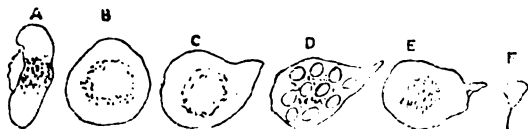


FIG. 2.—Drawings made from personal observation, August 7th, 1901. A—E shows changes that took place in a crescent body during an hour's observation.

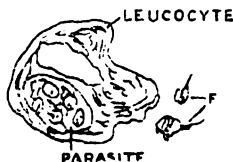


FIG. 3.—Same patient, August 8th, 1901. Personal observation. Malarial parasite before being picked up by leucocyte gave off (or out) two small active bodies, as shown in A, figs. 2 and 3. Those in the phagocyte seem to be identical in structure with those seen outside, fig. 2.



FIG. 4.—Crescent-derived sphere apparently segmenting. Picked up by leucocyte (fig. 4, D). Personal observation half-an-hour. Two of the bodies that were seen in (C, fig. 4) could not be made out in the phagocyte.

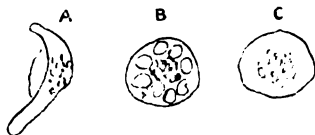


FIG. 5.—September 13th, 1901. Sphere that appeared as a segmenting body, but amœbulæ disappeared, just faded, and suggesting that they were probably vacuoles. (Personal observation.)

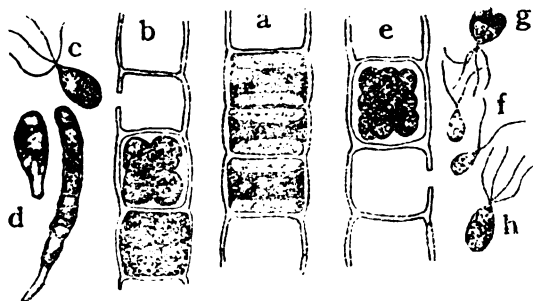


FIG. 6.—Ulothrix; a, vegetative filament; b, development of asexual zoospores; c, zoospore; d, sporelings; e, cell containing gametes; f, gametes; g, conjugation of gametes; h, sexually formed spore.—Davis, *Popular Science Monthly*, November, 1901.

In fig. 2 A will be seen two of these bodies very much like those in the phagocyte. They are about

one-eighth or one-tenth the size of a red blood-cell, comma-shaped, apparently made up of chromatin and protoplasm.

These bodies are actively motile, and contain oftentimes a granule of pigment. They are certainly not granules of any kind from leucocytes, nor are they Miller's "blood dust." I have repeatedly seen them escape from the crescent-derived spherical body soon after the parasites had begun to show signs of decay. I have tried to stain them, but so far have failed. Their origin and nature may be known, but to me they are not explained.

Davis (*Popular Science Monthly*, November, 1901), in speaking of the work of Klebs, says: "His studies on Ulothrix are interesting. The zoospores (fig. 5, B) are formed in varying numbers, but usually four or eight in a cell. They are relatively large structures with four cilia. The four ciliate zoospores are never sexual, and they develop new ulothrix filaments like their parent. This simple method of reproduction may be continued for many months, but at times the conditions are such that another form of swarm-spores appears. These elements are much smaller and have two cilia (fig. 4, B). They are gametes, and as a rule fuse readily with one another in pairs. If conjugation does not take place, the gametes settle down, and in certain instances have been observed slowly germinating; but they develop feeble plants."

Now what are the causes that make the plant produce asexual zoospores on the one hand and gametes on the other?

The problem thus resolves itself into an inquiry as to the precise environmental influences, the chemical and physical factors affecting the ulothrix filament, whether they are actually able to make the plants form zoospores or not according to certain conditions. It may be that these bodies which I have observed conjugate under proper environmental influences, and thus revert to the asexual cycle as indicated in the diagram.

Ewing ("Clinical Pathology of the Blood") has stained by the Noch and Romanovsky method a number of conjugating bodies of the malarial parasite. Thayer and others have called attention to this frequent conjugation of the amœbulæ of the plasmodium.

There is an interesting discussion of Dr. Ewing's paper in *Johns Hopkins Bulletin*, April, 1900. While it is pretty generally admitted that conjugation of the parasite occurs, I know of no suggestion that these small conjugating bodies are derived from sexual bodies which are returning to their previous method of development by sporulation. Why this departure from their usual course of development, except it be to continue their existence by conjugation, so as to give the most vigorous offspring obtainable under the circumstances?

Dr. Davis, in *Popular Science Monthly*, further says: "We may feel sure that sexual elements, gametes, have arisen from asexual reproductive cells with immediate relation to, and probably because of, certain environmental factors. In a general way these factors are known to be light, temperature, osmotic pressure and, most important of all, the

chemical nature of the environment, with especial reference to the kinds of food."

The period of latency in our cases has varied from a few days to one and a half years. It would seem, from a study of these cases, that when treatment is given the asexual amœbulæ, or parasites, are destroyed, leaving behind only those forms that are distinctly sexual. These bodies are very resistant to treatment (see history of cases). These sexual forms apparently produce no symptoms of their presence, but as soon as sporulation and conjugation of the sporozoites takes place, then segmentation follows. The rapidity of these changes depending entirely upon environment, and this environmental condition depends largely upon acclimatization, immunity, bodily vigour, &c. Hence the importance of making routine examinations of the blood, and if the parasites be present there should be the most energetic treatment begun and continued until every vestige of a parasite disappears. Then, in order to catch other bodies that may result from the segmentation of conjugate forms, quinine ought to be given at intervals ranging from six to twenty-seven days, and continued at these intervals for several months. In a more general way we may say that the duration of the latency depends upon the following, viz.:

(1) It may be that owing to a lack of development of the parasite in sufficient numbers there is not enough toxin liberated at a time to cause symptoms. That such substances of a toxic nature are produced by the parasites themselves, and liberated, or that the disintegration of the red blood-cells so alters the blood circulating through the body that toxic effects result most investigators admit.

Celli, while arguing strongly in favour of the toxins being generated by the parasites, says: "We have not been able to demonstrate pyrogenic toxin in the serum of the blood of those suffering from a malarial attack."

(2) The parasites may become attenuated to such a degree that they do not produce the amount and the kind of toxins to manifest symptoms of their presence, though they may be present in large numbers.

I have often seen cases where the symptoms in no wise seemed commensurate with the number of parasites observed in the specimen of blood. There are a number of examples of attenuation in the growth of bacteria.

(3) The individual may have acquired a certain immunity, so that he either prevents a sufficient development of the parasites that they do not liberate enough toxins to cause symptoms, or else this immunity has enabled the organism to become resistant to such large amounts of toxins that no symptoms are manifested.

(4) The attenuation, lack of development, and partial immunity may have been of such a character that the symptoms manifest themselves in an atypical way; or the presence of some disease associated with the malarial infection may so modify the symptoms of malarial fever that they become atypical. In these instances I would be disposed to speak of it as MASKED rather than latent.

Case I.—Simple tertian. Latent 7½ months; symptoms of recurrence. D. O., aged 12. Came to Gal-

veston from Paris, Texas, about November 1st. Had been having tertian chills for several months before coming to Galveston. Got them stopped. Had been in good health until May 15th, 1901, when he was taken with nausea and vomiting. Nausea continued for twenty-four hours. Blood examined the following day, and found to contain *tertian parasites*. Quinine was given, with complete relief of nausea and fever. Had a recurrence on the 26th day of May. Quinine was again given, and continued. Patient left the city on the 13th of June apparently cured.

Case II.—Simple tertian, becoming double tertian. Latent 8½ months. J. M. (c), aged 8. Admitted August 3rd, 1901. Has been in Galveston about three years, except about one month (September, 1900), he was at Chenango, Texas. Had tertian chills and fever a short time after leaving Chenango. Took medicine and got well. June 19th, 1901, chill and fever returned, and recurred every third day for about one week; since then he has had a chill every day. Tertian parasites one-half and full-grown, seen August 3rd, 1901.

Case III.—Tertian. Relapse after three weeks. Masked. F. M., aged 24. Admitted August 25th, 1900. Previous health good. Drinks beer and whisky. Labourer on farm at Dickinson, Texas. Took sick June 15th. Tertian chills for one week. Took treatment. Chill again after three weeks. No chill since August 15th, but has had fever every other day. Aching of bones and loss of appetite. Tertian parasites.

Case IV.—Double tertian, showing disposition to relapse. F. M., aged 35. Admitted October 18th, 1900. Malaria in Africa in 1892. Lasted six months. Contracted malarial fever in Houston, Texas. Chills began in July. Tertian. Took quinine, and would miss chills for two weeks. October 8th, chills and fever returned, becoming quotidian. Anorexia, headache, and pain in region of spleen. Spleen enlarged. Tertian parasites.

Case V.—Simple tertian. Latent probably 1½ years. J. W., aged 59. Admitted October 27th, 1900. In Galveston four weeks. Came from Dallas, Texas. Rheumatism five years ago. Had malarial fever two years ago, which lasted six months. Was apparently well. Two weeks ago chills returned. Tertian. Spleen enormously enlarged, extending 12 cm. below costal margin in mammary line and to median line of body. Tertian parasites.

Case VI.—Simple tertian. Latent five months. De L., aged 44. Admitted September 11th, 1901. Born in Mexico. In Texas five months. In Galveston most of the time. Since coming has not left the island. Disease dates back five months. Subject to intense headaches, worse during the past month. Distinct chill on the 12th of September. Tertian parasites.

Case VII.—Double tertian. Latent 5½ months. W. J. F., aged 45. Admitted April 4th, 1901. Has been in Galveston, Texas, twenty-four years. Had malarial fever August, 1900. Tertian. Recurrence about September 29th, 1900. The present attack began March 15th, 1901. Chill, fever and sweat every third day. About April 1st chills began coming every day. Two groups of tertian parasites were found.

Case VIII.—Simple tertian probably. Many relapses. F. A. H., aged 21, medical student. In Texas seven years. In June, 1895, one year after coming to Texas, began having tertian chills. These came every three weeks, then relapses began every 14th and 21st day. Still tertian. In May, 1896, chills returned, and kept up until Christmas after skipping several weeks. In 1897 chills came on in the spring and kept up during the year. 1898 at Ryan, Texas. Many cases of malaria there. Chills kept up until September, 1900. Came to Galveston November 1st, 1900, and chills returned again January, 1901. Again stopped them by quinine. April 1st and 2nd, chills. Took quinine. They now came every 7th to 14th day. May 3rd, 1901, tertian parasites.

Case IX.—Double tertian. Post-operative malaria. Latency five months. Mrs. W. B. P., Rosenberg, Texas, aged 34. Mother of five children. Has had chills and fever every year. Came to Galveston about October 1st, 1900. Soon after coming she consulted me for a severe attack of dysentery. Numerous amœbæ were found. No note of a blood examination was found. She was treated about ten days, and returned to Rosenberg in November. Was not very well. Suffered pain about body at different times, and felt exceedingly nervous. Had a severe chill on January 17th, 1901. Took large doses of quinine. Had three more third day chills. Had no other chills, though she suffered more or less pain all the time. Very nervous, great lassitude; poor appetite; loss of weight; sallow complexion. She returned to Galveston April 7th. April 10th I was consulted again. No history of chills or fever since January while at Rosenberg. Said she had tingling sensation in limbs, and was nervous and irritable. Poor appetite and no energy. Pain in lower part of abdomen. Examination showed laceration of perineum, lacerated cervix, and some prolapse of uterus. Uterus enlarged and tender. Temperature normal. May 6th chloroform was given, uterus curetted and cervix repaired. Her improvement was rapid. Three weeks later chloroform was given, and perineum repaired. Her recovery was not so rapid. Temperature ranged about 99° F. Removed stitches on the 12th day. Temperature normal. On June 30th she complained of a burning sensation of the skin and pain in joints. July 3rd I found she had a temperature of 101.5° F. She had had no chill or chilliness, and was surprised when I spoke of the fever. A blood examination showed two groups of the tertian parasite.

Case X.—Double tertian. Showing frequent relapses. H. Y., negro, aged 17. Admitted July 2nd, 1901. Health good previous to October 6th, 1900. Went from Galveston to Fort Bend County, on Brazos river, September 22nd. February 6th was taken with a chill, followed by fever and sweat. The chills recurred every third day until December 25th, 1900, although quinine was taken. Missed seven days, then chills recurred for several days. Quinine again. Stopped until April 18th, 1901, since which time they have recurred at irregular intervals, changing June 28th from tertian to double tertian. Half-grown and full-grown tertian, segmenting and flagellating parasites demonstrated on July 2nd, 1901.

Case XI.—Simple tertian. Relapses. Great resist-

ance to treatment. W. N., aged 25. Admitted October 4th, 1900. Drinks whisky and beer. Was at work on Brazos river. Went to Mineral Wells and remained there for some time previous to taking sick. Was taken sick May 15th, 1900. Chills and fever every third day up to July 12th. Since then no chills, but has had fever at different times. Often goes three weeks free of fever, then a recurrence of fever every day. Has had fever now about three days, aching of bones and headache. Spleen very much enlarged. During the three days he was in hospital he had no rise of temperature, nor did he have a chill. Tertian parasites.

Case XII.—Æstivo-autumnal. Latent for about seven weeks. Post-operative malaria. Mrs. O. B., aged 26. Admitted September 6th, 1901. Been in Galveston six years. Born in Texas. Typhoid fever at 14. Very strong and healthy when a girl. Menstruation at 14 normal. Married at 16. One child at 17, and one at 20. Menstruation painful and irregular since last child. Severe pain in back and both iliac regions. No chills or fever since the latter part of July, 1901. Complained of aching all over for more than a month. Appetite poor, drowsy, no energy. September 15th, 1901: æstivo-autumnal ring-forms in blood, no leucocytosis. September 16th, 1901: Many crescents and ring-forms. No leucocytosis. Urine normal. Operation on the 7th. One tube and ovary removed by Dr. Ruhl. Dr. J. H. Ruhl, to whom I am indebted for the report of this case, says that the temperature was normal for ten days before operation.

Case XIII.—Æstivo-autumnal, showing latency and resistance to treatment. G. B., aged 42. Admitted September 9th, 1901. Has lived in Galveston twelve years. Went to Denison, Texas, in March, and remained there until June. Went to Beaumont July 7th, and remained there until August 24th. Returned to Galveston September 3rd. Previous health good. Took sick with fever September 6th, followed by sweats three hours later. No chill at any time. Backache and pain in hips. Æstivo-autumnal parasites. Small intracorporeal æstivo-autumnal bodies on the 9th, 11th, and 16th. Crescent bodies seen first on September 16th. Still present on October 3rd, when he left the hospital.

Case XIV.—Æstivo-autumnal. Latent one year, and relapse again after one month. J. A., aged 29. Admitted September 15th, 1901. Previous residence Cleveland, Texas. Left there and came to Galveston May 20th. Had rheumatism three years ago. Had malaria fever in July, 1900. Got well apparently. Had another chill July 1st, 1901. Took quinine, and was well for one month. August 10th, chills returned, occurring every day. High fever followed by sweating August 16th. Blood examination showed æstivo-autumnal parasites.

Case XV.—Æstivo-autumnal. Latency fifty-two days. D. C., aged 34. Admitted September 19th, 1901. Went to Beaumont about July 10th, 1901. After being there two weeks, he began to have diarrhoea and fever. Took quinine and got all right. Came back to Galveston in August. September 15th was taken with a chill, followed by fever. Æstivo-autumnal parasites were demonstrated on the 16th, 17th, 18th, and 19th.

Case XVI.—Æstivo-autumnal. Latent six months. Another relapse. Z. A., aged 35. Admitted September 20th, 1901. Was in Dickinson during February, March and April. Came to Galveston May 20th, and remained until July 20th, when he went to Lampasas, Texas. While there in the first part of August had chills and fever for eight days. Returned to Galveston August 8th. Took medicine and remained well until September 6th, when he began having fever, pains through body, and once in a while chilly sensations. Parasite of æstivo-autumnal type was found on September 21st, but no crescents or ovoids. Crescents were present on September 27th. Patient left hospital September 29th, feeling well. Temperature had never reached 98.6° F. until the day of leaving, although under constant influence of quinine.

Case XVII.—Æstivo-autumnal. Masked. Typhoid fever. L. M., aged 8. Admitted March 3rd, 1901. In Galveston one year. Was exposed to bad weather the last day of December, 1900. Remained in wet clothes at school all day. Had severe chill followed by fever. Was better next day, and returned to school. After two days was not well, and went to bed. Was in bed only a part of the time. Finally, about three weeks ago was put to bed. A doctor was called and made a diagnosis of relapse of typhoid fever. Patient has been having fever, headache, gastric pains, and a slight diarrhoea. Spleen much enlarged and tender. Abdomen distended. Crescents. Widal reaction "partial." Red cells 3,071,000. White 28,200. Diagnosis of typhoid and malaria.

Case XVIII.—Æstivo-autumnal. Latent 6½ months. J. C., age 36. Admitted April 15th, 1901. In Galveston four years. Probably contracted malaria in western part of Galveston. Was in the hospital in July, 1900, suffering from æstivo-autumnal malaria.¹ Went out free of symptoms, though parasites were still present. Chills began February 15th, 1901. Fever and sweat every other day. Took chill tonic and missed until March 19th, when from that time on they became quotidian. April 15th, intra- and extracorpuseular bodies, ovoids, crescents and flagellated parasites. Note.—No crescents or ovoids were seen during his stay in hospital in July, 1900.

Case XIX.—Æstivo-autumnal parasites present eleven days after temperature reached normal. P. Y., aged 25. Admitted July 6th, 1901. About June 6th went to Cleveland, Texas. Worked in river bottom and drank water from shallow well. Took sick a short time after going there. Pain in head and knees. Vomiting. Spleen enlarged. Small bodies of æstivo-autumnal parasites found on July 7th. Parasites were demonstrated in blood constantly, although temperature was normal from July 18th to the 29th, when he left the hospital. A large number of ovoids and crescents were seen on this date.

Case XX.—Æstivo-autumnal. Incubation apparently five months. S. L., aged 26. Admitted July 4th, 1901. Worked in Cleveland, Texas, five months ago, and drank river water. No previous diseases. June 24th began to have abdominal pain, headache, fever, chilli-

ness, diarrhoea and vomiting. Has been very sick since. Spleen enlarged. Æstivo-autumnal parasites.

Case XXI.—Æstivo-autumnal. Incubation twenty-four days. Relapse. J. K., aged 40. Admitted August 16th, 1901. Had typhoid and malarial fever about fifteen years ago. Rheumatism ten years ago. Health since then has been good. Has been in Galveston about seven years. Has occasionally visited other places in the State. June 13th patient went to Cleveland, Texas. Slept on the bank of the river. Most of the men working there had malarial fever. July 7th had a chill, fever and sweat. Took medicine. Had tertian paroxysms for about one week; then chill came on every day for two or three days. After this, had no more chills until August 22nd. Blood examined on the day of admission showed no malarial parasites. On the 24th blood was examined again, and showed the ring-shaped æstivo-autumnal bodies. Slight chill on the 25th.

Case XXII.—Æstivo-autumnal. Showing parasites present with a normal temperature. M. J., aged 29. Admitted August 17th, 1901. Rheumatism at different times during the past five years. June 19th, patient went to Bellville, Texas. Had chills and fever while there. Came back to Galveston the latter part of August, 1900. Had chills and fever off and on during the winter. They would stop while taking quinine, but would soon return. About May 25th, 1901, went to work on Trinity River. Was there until July 15th, when he went to Beaumont Hospital for the trouble. Was there until August 16th, when he came to Galveston.

Blood examination on August 17th; intracorpuseular bodies and crescents. Blood examined at intervals of two days showed parasites present each time. September 23rd parasites present: intracorpuseular bodies of æstivo-autumnal fever. Temperature had been normal for thirteen days.

Case XXIII.—Æstivo-autumnal. Latent 2½ months. R. J. K., aged 45. Admitted September 23rd, 1901. Syphilis five years ago. Otherwise health good. Went to Beaumont first part of April, 1901. Was taken with dizziness in head in early part of May. Two weeks later had a distinct chill and fever. Came every third day. Took medicine and felt well. Left Beaumont and returned to Galveston latter part of June. Continued well until September 12th; began to have pains through body, headache, and general malaise. On the 19th, was taken with a decided chill followed by fever. Fever continues. Chill again on the 22nd, 23rd, and 24th. Since then he has had no chill, nor has there been a rise of temperature above normal. Small æstivo-autumnal parasites present on the 23rd and 24th. Ovoids and crescents seen first on September 25th, and again on the 27th. Intracorpuseular ring bodies and crescents abundant on the 27th.

[Owing to lack of space the sketch maps and the temperature charts illustrating this interesting paper could not be produced.—Ed. J. T. M.]

¹ Case XVIII. was previously reported by the writer ("Malarial Nephritis," *Trans. U. S. Med. Association*, 1901).

PRINCIPLES DETERMINING THE GEOGRAPHICAL DISTRIBUTION OF DISEASE.

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THE study of disease in relation to surrounding conditions has at all times been a subject of earnest inquiry, and of this we have ample evidence throughout medical literature, from the early books of Hippocrates, *περί ἀέρων, ὕδατων, τόπων*, to Hirsch's monumental work on geographical and historical pathology. However, notwithstanding the mass of materials collected, especially with a view to the elucidation of the etiology of disease, the laws which control geographical pathology have not yet been formulated, or at any rate they have not been settled in accordance with recent science. Indeed, the principles which regulate the distribution of disease, and determine the rise and fall of epidemics, could not have been conceived without the light of the evolution theory, and a study of the distribution of species founded on plant and animal ecology.

To thoroughly apprehend the laws which govern the geographical distribution of disease, it is necessary to have a clear notion of the nature of disease. Unfortunately, whilst the etiology of certain maladies has been satisfactorily worked out, that of many others is still shrouded in obscurity.

Undoubtedly, the greatest advance ever achieved in medicine has been the establishment of the parasitic theory. The belief in a living cause or *contagium vivum* is very old, but it is only quite recently that it has been demonstrated. However, notwithstanding the discovery of specific organisms in a number of widely different disorders, formerly attributed to astrological, meteorological, or chemical causes, the all-important part played by parasitism in disease causation does not seem to have been fully grasped. It is true that some diseases, such as measles, scarlet fever, small-pox, yellow fever, have long been classed among the parasitic diseases, merely on account of analogy with similar disorders, the parasitic nature of which has been fully demonstrated; but the analogy has not been extended beyond a certain group of diseases called "infectious diseases."

In medicine, as in all other branches of knowledge, there has always been a strong aversion to relinquish the old orthodox theories, and, indeed, far more energy is spent in opposing a new idea than in advancing it. One of the diseases earliest acknowledged to be of parasitic origin was scabies. The Arab physicians of the twelfth century had a clear notion of its etiology, and, in the writings of Avenzoar we find the *Sarcoptes* clearly mentioned; but, notwithstanding the repeated observations of numerous naturalists, the psoric acarus was utterly ignored by medical men, who continued to explain the disease by the humoral theories up to the year 1834, when a Corsican student, Francesco Renuccio, hearing the existence of the acarus denied at the Saint-Louis Hospital in Paris, proposed to show it forthwith, and extracted it from the epidermis of a patient with the point of a needle, as he had seen the peasant women of his country do many a time.

Almost every disease has a similar history. A recent example is that of the opposition offered to the discovery of the hæmamœbiæ of the intermittent fevers, and to the demonstration that these parasites are propagated by certain species of mosquitoes which subserve them as definitive hosts.

The classification of diseases in modern text-books is very confusing and misleading. Whilst two sections are usually set apart, one for diseases due to parasitic worms, insects and mites, and the other for certain diseases called "infectious," and now known, or believed to be caused by parasitic protozoa, fungi, or bacteria, all other diseases are still grouped in the old way, according to the several organs or systems of which the body is composed.

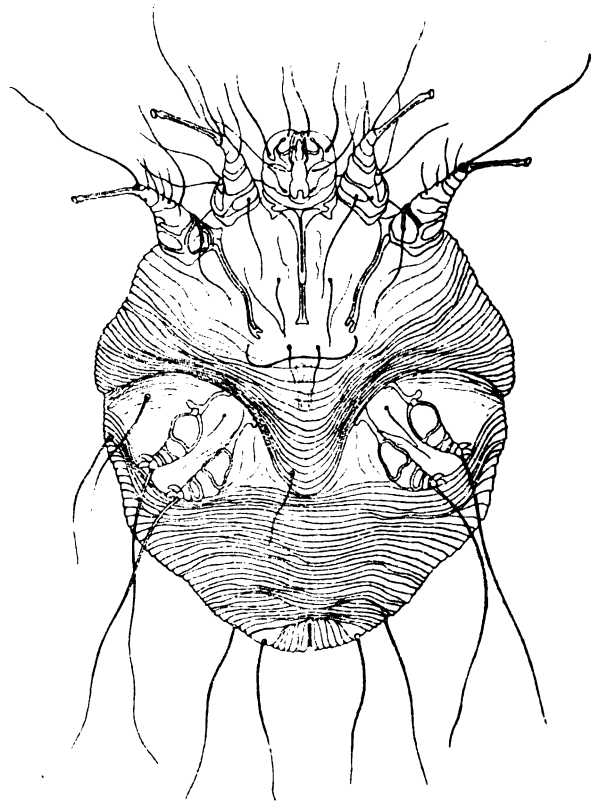


FIG. 1.—*Sarcoptes scabiei*, ovigerous female, seen on the ventral surface. Magnified 160 diameters.

If diseases were arranged according to their etiological factors, much confusing repetition would be avoided, the absurdity of certain old theories would be evident, and the student would acquire a more lucid and definite idea of the nature and mechanism of disease. After a cursory chapter on the injuries that may arise from lightning, fire, frost, or poisons, from the weapons of man, the bite of wild animals, or the stroke of venomous snakes, the diseases caused by the parasitism of insects, acari, leeches, nematodes, cestodes, trematodes, protozoa, fungi and bacteria, should follow successively in a natural biological order. Of course, it would be difficult just now to place satisfactorily those diseases of obscure etiology which still bear the old labels of faulty metabolism,

alcoholic excess, or exposure to cold and heat. But we should not forget that all the diseases now known to be of parasitic origin were, at one time, erroneously ascribed to these very same causes.

For generations physicians have considered cold to be the *prima causa* of pneumonia and pleurisy. The highest incidence of pneumonia is in the winter and spring months, its occurrence after a wetting, or a chill due to some unusual exposure, its constant and severe initial rigor, seemed very obvious indications of the correctness of such a theory. Now we know that lobar pneumonia is due to *Micrococcus lanceolatus*, and that broncho-pneumonia and pleurisy may be caused by various parasites, such as *Bacillus tuberculosis*, *Micrococcus lanceolatus*, or *Streptococcus pyogenes*.

Rheumatic fever is another disease which was believed to be due essentially to cold. Now it is classed almost unanimously amongst the infectious diseases, although no positive proof has as yet been offered of the constant association of any special micro-organism with the disease.

It would occupy too much space to enumerate all the diseases which have been attributed to cold; but, in order to prove the absurdity of this theory, I will mention that even in such a progressive text-book as Osler's "Principles and Practice of Medicine," apart from frost-bites and chilblains, cold is still given as the universal cause of arthritis deformans, chronic rheumatism, acute tonsillitis, catarrhal jaundice, rheumatic peritonitis, coryza, acute laryngitis, acute bronchitis, idiopathic pericarditis, acute endocarditis, Bright's disease, locomotor ataxia, progressive muscular atrophy, infantile paralysis, ataxic paraplegia, acute myelitis, neuritis and neuralgia.

It may, perhaps, seem unwise to question the universally accepted etiology of such diseases as scurvy, gout, or diabetes; but there are numerous conditions in the natural history of these diseases which cannot be explained by the prevailing chemical theories, and which are distinctly suggestive of parasitism.

I need not discuss the etiology of scurvy, because its specific nature has already been very ably advanced. In parts of Russia scurvy is endemic, assuming at certain seasons epidemic proportions, and Russian authorities are almost unanimous in regarding it as infectious. The infectious origin of diabetes has also been suggested, and chiefly on the grounds of conjugal diabetes.

I will mention certain facts which, to my mind, suggest the parasitic nature of gout. Gout has a wide geographical distribution, but it is absent in many places. It is quite unknown in Lapland, in Iceland, in the Farøe Islands, in Ceylon, in New Zealand, and the Hawaiian Islands (Hirsch). It seems to be absent from the greater part of the African Continent and from Australia (Hirsch). In America, it is a comparatively rare disease, but it has become more common of recent years in the United States (Osler). It is more frequent in England than in Germany, but although rare in Leipzig it is rather common in Bavaria (Strümpell). The prevalence of gout differs considerably at various places within its endemic areas, quite irrespective of the conditions believed to be most favourable to its occurrence: it has completely dis-

appeared from certain districts without any apparent reason; in others, it has greatly fluctuated at different times. In its typical form, gout is a paroxysmal disease. In the interval, the patient feels quite well, and, as Aretæus remarked, "He may have won the race at the Olympian games." A patient may have three or four attacks in a year, or the fit, as it is called, may not recur for several years. The characteristic uratic deposits of gout do not occur simultaneously in all the joints, but successively, and each paroxysm is connected with a peculiar inflammatory process in one or other joint, and the subsequent deposit of crystals in the affected joint.

Excess of uric acid in the blood is by no means peculiar to gout; it may occur in leukaemia, chlorosis, and other diseases which do not present any of the other symptoms peculiar to gout. Therefore, uric acid cannot be regarded as the cause of gout any more than lactic acid that of rheumatic fever.

Gout, or disorders indistinguishable from the gout of man, are met with in hogs, parrots, fowls, ostriches, and reptiles, all of which cannot be said to have been influenced by the "evils of civilisation," to which Balfour and others ascribe the "gouty diathesis."

The great Boerhaave believed gout to be a communicable disease, and Van Swieten, in his commentaries on the "Aphorisms," adduces in evidence that wives who had tended their gouty husbands day and night were ultimately attacked by the disease. The communicability of diseases varies greatly with the different conditions necessary to their propagation. Certainly, one would not compare the infectiousness of gout to that of dengue or influenza, but to that of diseases long considered as non-infectious, such as leprosy and tuberculosis.

Great stress has been laid at all times, on the hereditary transmission of gout. But, in the light of modern knowledge, inheritance of disease spells infection. We do not believe any longer that syphilis, tuberculosis, or leprosy are hereditary diseases in the strict scientific sense of the word "hereditary," which means the development of characters existing potentially within the protoplasm of the first formative units, but that they are transmitted to the foetus during the period of gestation. Hauser, in his excellent study of hereditary tuberculosis, reached the conclusion that there were really no satisfactory instances on record of the transmission of tuberculosis from parent to child, except in a few cases in which the mother was suffering from miliary tuberculosis during pregnancy. Experimentation has clearly proved that a father suffering from tuberculosis does not transmit the disease to his offspring by a healthy mother. Authors have often remarked upon the frequency with which gout affecting grand-parents would skip one generation to reappear in the grandchildren. A similar fact is noticed in tuberculosis and other diseases known to be of parasitic origin. It may be explained by an inherited immunity sufficient to protect the children, but not sufficiently permanent to protect the second generation.

At one time, when nothing was known of their etiology, intestinal worms were likewise believed to be hereditary, and the old physicians spoke of a

"verminous diathesis" just as gravely as we now talk of a "gouty diathesis."

Another very interesting disease which can be better explained by parasitism than in any other way, is calculosis. The geographical distribution of urinary calculus is somewhat known in a general way. It is very wide but most unequal. In certain places the disease is very common, in others it is quite unknown. Thus, it is almost unknown in Ireland, while it is very common in Norfolk, where it has been ascribed to the use of Norfolk dumplings. At one time, calculosis was ascribed to a cold and damp climate, but its prevalence in Syria, Persia, India and China, and its extreme rarity in Norway, Sweden and Alaska, were opposed to such a theory. Its supposed relation to chalk soil was likewise contradicted by the facts of geographical distribution, and physicians were obliged to excogitate a lithic diathesis analogous to that of gout.

In favour of the parasitic theory we have already a number of facts. Thus, calculosis is a special feature of Bilharzia disease, and the nucleus of the calculi is usually formed by the ova of the parasite. In hepatic

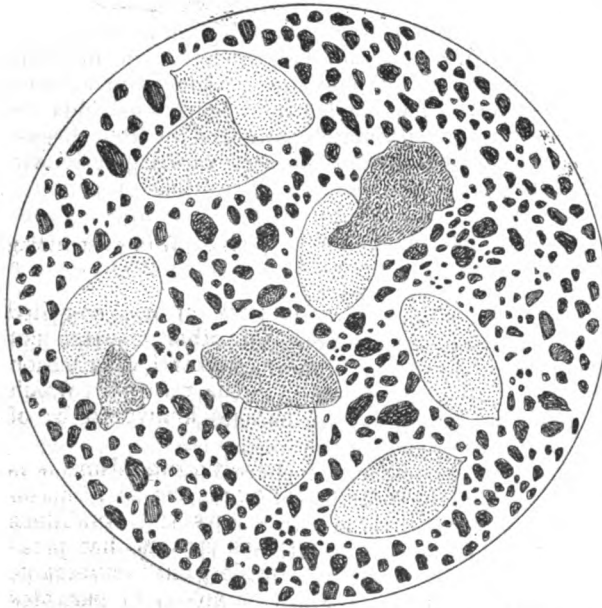


FIG. 2.—Section through nucleus of urinary calculus containing ova of *Schistosoma hematobium*.

distomiasis biliary calculi are frequently formed, and they may contain one or more flukes, as was observed by Simonds and Brousson. Recently Welch and others have demonstrated the presence of micro-organisms in the centre of gall-stones, and thus explained the frequent association of cholelithiasis with typhoid and other specific fevers. Gilbert and Fournier succeeded in producing gall-stones by injecting micro-organisms into the gall-bladder of animals. Calculosis is a process very similar to that of calcification which occurs within the tissues round encysted parasites, such as trichinæ and bladder-worms.

I need hardly state that in recent years parasitism

has completely revolutionised the etiology of skin diseases. In neurology, parasitism is perhaps less apparent, but surely no one can fail to perceive that the recognition of the specific agents of tuberculosis, leprosy and syphilis, in neuro-pathology, points to a radical modification of our conceptions of the etiology of nervous diseases.

Another great group of diseases which must be mentioned is that of tumours. Already the granulomata have been ascribed to specific parasites, such as the fungus of actinomycosis, or the bacilli of glanders and tuberculosis, and recent investigations point to certain protozoa as the specific organisms of carcinomata and sarcoma. But the etiology of the other neoplasia is still hampered by untenable theories, such as Cohnheim's theory of embryonic residues.

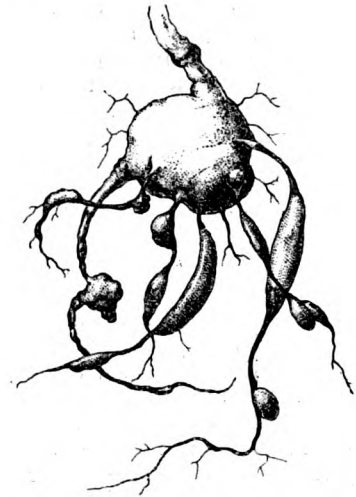


FIG. 3.—Root of young turnip with clubs. One-half natural size.

The old theories would have been abandoned sooner, perhaps, if the causation of tumours had been studied in the wider field of comparative pathology. In plants we meet with analogous growths called *cecidia* or galls, of which the "oak-apples" and "witches' brooms" are well-known examples. Botanists have already classified more than 1,600 different gall-structures, but it is not long since their etiology was discovered. At one time, galls were believed to be caused by elves and witches, later they were ascribed to unusual meteorological conditions; now, at last, they are known to be caused by parasites, which may be fungi, mites or insects. Galls vary greatly in size, shape and structure; some are solid, others hollow, like cystic tumours; some are rapidly deadly to the host-plant, others are harmless, and may fall off in autumn, like ripe fruit. Some are limited to small leaf-areas, and are formed by hypertrophied epidermal cells, which grow out exactly like warts; others modify whole branches, and alter the internal structure as well as the outward appearance of the host-plant.

The same species of parasite produces very similar, but slightly different galls, on different plants, while different parasites produce very dissimilar galls on the same plant. Some oaks, for instance, may bear as

many as thirty different forms of gall produced by as many kinds of gall-wasps.

Naturalists have been greatly puzzled as to the actual process of gall formation. Now, they believe that the active cell-division which produces the gall is incited by some kind of substance excreted by the parasite. In galls produced by insects, the eggs deposited in the plant tissue, or attached to it, are incapable of inciting gall formation. The formation of the *cecidium* commences after the hatching of the larva, and invariably ceases if the animal dies. The abnormal growth is probably an effort to repair injury. It is well known that the largest and best looking pears in orchards in the early summer are often swarming with larvæ. The larvæ stimulate the growth of the young pears, causing a spurious appearance of unusual health and vigour. Parasitic fungi, which attack leaves or roots, usually cause great distention of the cells they affect. In the club-root disease of turnips, commonly called "fingers-and-toes," the swellings are due to the enormous enlargement of the cells containing the parasite (*Plasmodiophora brassicæ*).

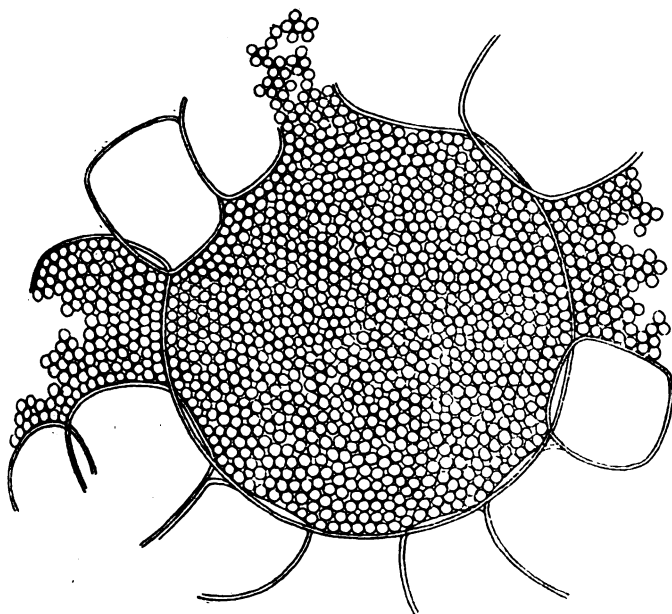


FIG. 4.—Spores of *Plasmodiophora brassicæ*, Wor., seen within the cells of turnip-root. Enlarged 400 diameters.

The comparison between the gall-structures of plants and animal tumours is not so far-fetched as might appear at first sight; anyhow, we should not forget that some of John Hunter's best work upon the diseases of bones was illustrated by the similar changes he met with in diseased tree-branches and twigs.

With a view to the etiology of neoplasia in animal pathology, it is important to note that some of the gall-producing fungi have two alternative stages of development. Each one of these two stages is spent on a different host; is characterised by a difference in the nature of the spore-producing organs; and induces

a very different kind of hypertrophy in the respective host-plant.

The importance of parasitism in the causation of disease becomes more and more apparent the wider the field we survey. Recent work in tropical medicine has added numerous examples, and has prompted the re-examination of analogous diseases at home. The study of the diseases of animals and plants has evidenced even more forcibly the pathogenic rôle of parasitism, and has overthrown a number of speculations which seemed reasonable enough when limited to human pathology, but which appear absurdly erroneous when extended to the same or analogous diseases in animals and plants. For example, alcoholism as the cause of certain diseases common to men and cattle!

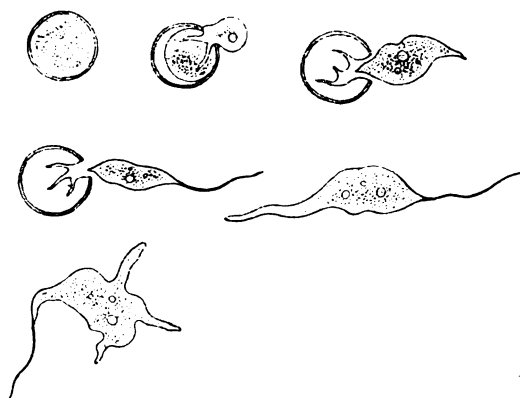


FIG. 5.—Spores of *Plasmodiophora brassicæ*, Wor., germinating and producing amoeba-like zoospores.

Whatever be the true causation of the so-called "constitutional diseases" and of other diseases not assigned to parasitism, their geographical distribution seems to be controlled by the same principles which govern the distribution of diseases known to be of parasitic origin.

If diseases are caused by parasitic organisms it is evident that to understand the origin and distribution of diseases we must study the laws and conditions of parasitism. At one time it was thought that parasites had always existed as parasites, but innumerable facts in the morphology and embryology of parasites prove that the parasitic habit is gradually acquired, and that all parasites must have evolved from non-parasitic forms. A number of parasites are strictly confined to certain plants or animals as hosts; it is therefore evident that they must have originated after the occurrence of the host-species either by adaptation from free-living forms or from parasitic species on other hosts; and following this back to its origin, we must ultimately arrive at a free form as the source. In many cases the line of evolution is quite apparent; as, for instance, the gradation between comparatively free and fixed bird-lice (*Mallophaga*).

An example which shows how parasitic habits may be acquired, is that of the Kea (*Nestor notabilis*), one of the long-beaked parrots peculiar to New Zealand. Before the introduction of sheep in New Zealand, the Kea fed on the juices of plants, but lately it has

become a sheep-killer. In 1868, it was noticed that the Kea was in the habit of visiting the carcasses of sheep, which were hung up for consumption, and eating the fat round the kidneys. The Kea has now become a formidable foe to the flocks of New Zealand, for it attacks the living sheep, perches on their backs, tears away the skin and digs out the kidney fat, thus causing their death.

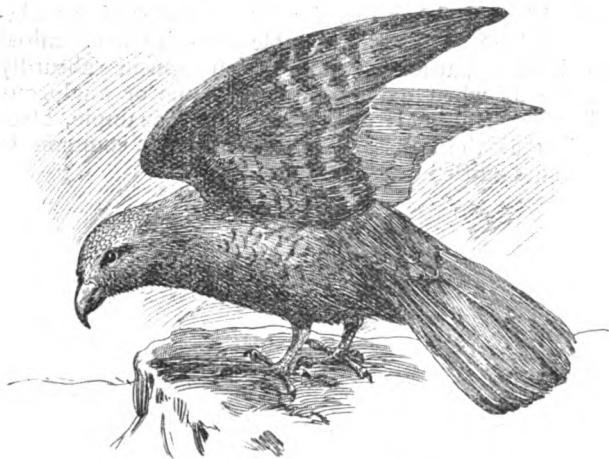


FIG. 6.—*Nestor notabilis*.

The natural tendency of an animal once started in the direction of parasitism is to become more and more parasitic in habit, and with this habit the structure of its body will gradually become modified. The modification is usually considered a degeneration, because it results in the loss or atrophy of certain structures which have become useless in a sedentary, non-competitive parasitic life; but it really is a limitation in certain directions, with a progressive elaboration in others. The structure of an animal is as inevitably associated with its functions and adaptations as the state of HO_2 with temperature.

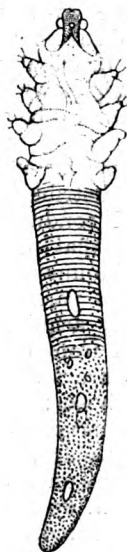


FIG. 7.—*Demodex folliculorum*, var. *hominis*.

Many parasites have become so greatly simplified that they present a very different appearance from those animals with which we know them to be closely related. Thus the linguatulæ, which belong to the class of the aracmida, were believed to be worms, until the study of their embryology placed them amongst the arthropoda. *Demodex folliculorum*, found in the skin of the human face, is a degenerate spider; its eight legs have become mere stumps, and the body is elongated like that of a worm. A striking example of modification due to parasitism is that of a crustacean, called *sacculina*. The young *sacculina* is an active, free-swimming larva, much like those of other shrimp-like animals. But the adult which lives as a parasite on the abdomen of hermit-crabs, is a mere sac filled with eggs, and provided with

delicate root-like processes, which penetrate the body of the crab host, and absorb nutriment exactly like the *haustoria* or roots of parasitic plants.

As an example of the gradual reduction of organs through parasitic habits, may be mentioned that of the wings in forest flies. *Hippobosca equina* which infests horses and oxen, has fully-developed wings; another species *Stenopteryx hirudinis*, which occurs on swallows, has narrow sickle-shaped wings scarcely fitted for flight. A third species *Lipoptena cervi*,

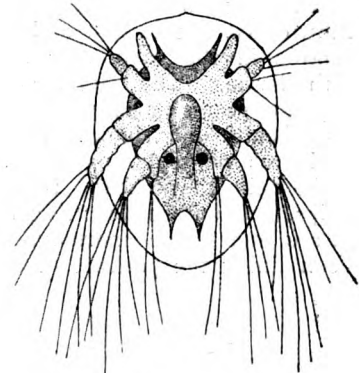


FIG. 8.—Larva of *Sacculina*.

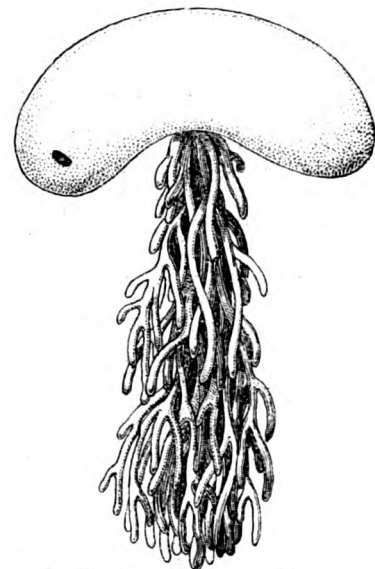


FIG. 9.—Adult *Sacculina* (female).

which is found on deer, is provided with wings upon issuing from the pupa case; but soon after settling on its host it drops them, by fracturing them at the base. A fourth species, the so-called sheep-tick (*Melophagus ovinus*), is entirely wingless from its birth. We thus get in this family a series of forms starting with the fully-winged horse-fly, and leading through the swallow-fly with reduced wings, and the deer-fly which can cast its wings, to the sheep-fly, which has entirely lost these organs.

Many parasites have become so specialised, so greatly modified and changed, in order to adapt them-

LONDON SCHOOL OF TROPICAL MEDICINE.

8th Session, Jan.-March, 1902.



J. R. Delnere, J. Maye, Robert (Laboratory Assistant), G. E. Brooke, A. T. L. Fox, J. A. Blaney, T. A. Dowse, H. E. Wareham,
Dr. W. G. Ross, P. A. Nightingale, Mr. K. W. Goadby (Lecturer), T. S. Kerr, Dr. Manson (Lecturer), Dr. Sambon (Lecturer), C. W. Seaton, R. E. G. Thilke,
Absent:—A. Fell, E. W. Lewis, J. Dodd, W. F. Manners, M. Macnicol, H. E. Wareham, W. P. Medtrum, H. E. Mann.

selves to certain hosts and to certain conditions of life, that they have become absolutely dependent upon such hosts and the presence of such conditions for their existence.

Sometimes the limitation to certain hosts is not associated with any apparent modification, as is the case with many insects which depend exclusively on certain food-plants.

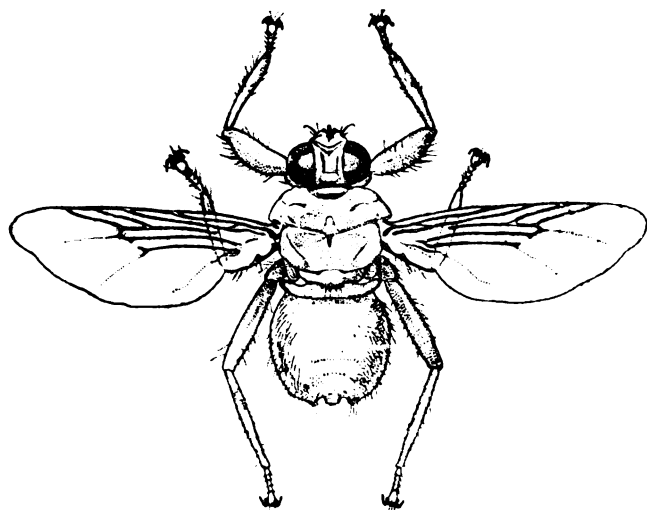


FIG. 10.—*Hippobosca equina*. Magnified 4 diameters.

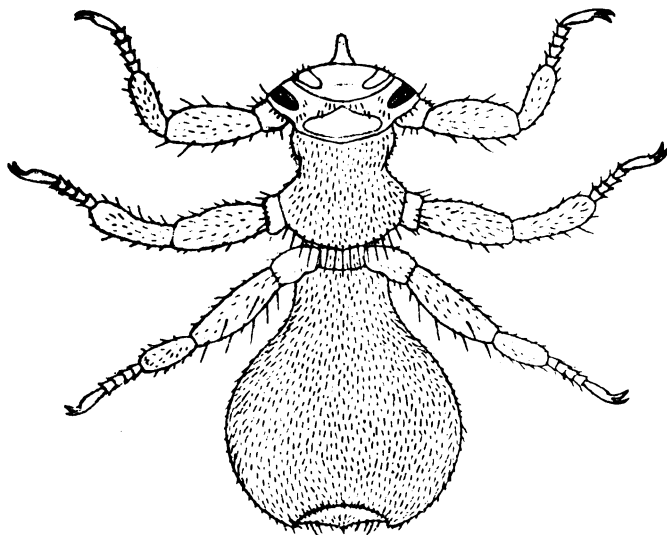


FIG. 11.—*Melophagus ovinus*. Magnified 10 diameters.

An example, which shows how such limitation may arise, is that of the Stem Eel-worm (*Tylenchus devastatrix*). This nematode lives and reproduces in various cultivated plants, such as rye, oats, stored onions, hyacinths, buck-wheat, potatoes, and clover, and in wild plants such as *Poa annua*, *Anthoxanthum odoratum*, *Dipsacus silvestris*, and *Polygonum persicaria*, but not to the same extent in all. However, Eel-worms, of which the progenitors have developed for many years exclusively in rye and buck-wheat, are

not easily transferred to another kind of plant, or at any rate, they do not multiply vigorously there.

An interesting example of the dependence of species on species is that of *Vedalia cardinalis*. A few years ago a pest called the cottony-cushion scale (*Icerya purchasi*) was introduced into California from Australia on young orange trees. This pest soon increased to such an extent that it threatened to completely destroy the great orange orchards of California. Artificial remedies having proved useless, a well-known entomologist, Dr. C. V. Riley, was sent to Australia to find out if this scale insect had not some special

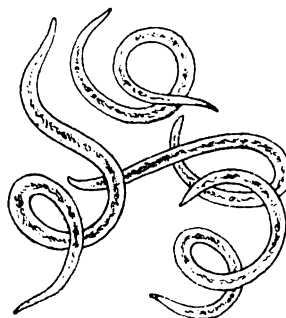


FIG. 12.—*Tylenchus devastatrix*. Magnified 200 diameters.

natural enemy in its native country. He found that in Australia a certain species of ladybird (*Vedalia cardinalis*) attacked and fed on the cottony-cushion scales and kept them in check. Some of these beetles were brought to California and released in a scale-infected orchard. Finding plenty of food the *Vedalias* thrived, and became so numerous and so widely distributed that the scales began to diminish perceptibly, and in a few years were almost wiped out. But the disappearance of the scales was followed by that of the *vedaliae*, and it was then discovered that these *coccinellidae* fed on cottony-cushion scales, and could not live without the latter. With the disappearance of the predaceous lady-birds, the scales began to increase again in various parts of the State; and now, in order to have a stock of *vedaliae* on hand in California, it is necessary to keep some colonies of the cottony-cushion scale to serve as food.

(To be continued.)

MOSQUITO LARVÆ AND MALARIA.—In the *Archives de Médecine Navale*, September and October, Paris, 1901, Dr. Palusne de Champeaux, from experiences gained during two military expeditions in Curamance, a country lying on the left bank of the Gambia, states: that the anopheles mosquitoes may cause malarial infection by their bite, and also that by drinking stagnant waters containing anopheles larvæ may the disease be spread. On what grounds Dr. de Champeaux arrives at the latter conclusion is not stated. He is averse to the administration of quinine as a prophylactic against malaria when no prodromal symptoms are present. It would be interesting to know why quinine prophylaxis is objected to and what are the prodromal symptoms which betoken the encroachment of the disease.

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THE Journal of Tropical Medicine

MARCH 15, 1902.

PRINCIPLES DETERMINING THE GEOGRAPHICAL DISTRIBUTION OF DISEASE.

We feel sure that the scientific appreciation of the new principles which Dr. Sambon has so kindly, and with so much originality and wealth of illustration, expounded, would contribute more to the prevention of disease than the blind, groping, very expensive, and, too often absolutely futile, efforts our sanitary authorities are wont to indulge in, more especially in tropical countries. We commend Dr. Sambon's paper to all interested in the philosophical study of disease and practice of medicine.

Some minds would have us draw a sharp line between the practical and the scientific in medicine; they would have us believe that the two are incompatible; that a scientific physician or surgeon cannot possibly be a practical physi-

cian or surgeon. Indeed, it is not unusual to attempt the detraction of a brother practitioner by alleging or hinting that although he may be expert with his microscope or test tube, even on that very account, he cannot prescribe for or treat successfully ordinary diseases. Exactly the same feeling exists in some minds as regards the sanitarian; that the man learned in germs cannot possibly know much about the operation of a patent water-closet. This attitude of mind is a very wrong and very foolish one. The mind that can grasp the principles of a science is just as likely, nay more likely than any other, to be able to apply that science. These remarks are elicited by the interesting paper by Dr. Sambon on the "Principles determining the Geographical Distribution of Disease," the first instalment of which appears in our present issue.

Obituary.

DR. PATRICK THURBURN MANSON,
M.B.LOND.

On Saturday, March 15th, news reached London from Christmas Island that Dr. Patrick Thurburn Manson, M.B.Lond., eldest son of Dr. Patrick Manson, C.M.G., F.R.S., met with a fatal accident shortly after arrival at Christmas Island.

We have only time, before going to press, to mention the fact, and to add our sincere sympathy with his parents and relations in their bereavement.

It is but two months since Dr. Thurburn Manson left London for Christmas Island, off the coast of Java, to take part in the scientific investigation of Beri-beri.

After a brilliant career as a student at Guy's Hospital he graduated M.B.Lond., and subsequent to graduation prosecuted his pathological studies at Aberdeen, and tropical diseases at the School of Tropical Medicine, London.

He left London full of enthusiasm for the work before him, and it is sad to think that so promising a career has been so suddenly closed.

A more complete account of his work and career will be given in our next issue.

News and Notes.

CHOLERA IN CANTON.—A severe outbreak of cholera is reported to be raging in Canton. So far Hong Kong has escaped infection, but the traffic between the two ports is so large and so constant that a spread of the disease to Hong Kong is to be dreaded.

Review.

THE MEDICAL ANNUAL, 1902. Bristol: John Wright and Co.

This useful publication has reached its twentieth year of issue. We congratulate Messrs. Wright and Co. upon their success, and we are sure all members of the medical profession thank them for one of the most highly appreciated of our medical publications. The present issue is equal to, and we think is fairly entitled to be considered an improvement upon, any of its predecessors. The work is profusely illustrated, and diagrams and charts help to render the descriptions of diseases interesting and educative.

Part I., "The Dictionary of *Materia Medica* and *Therapeutics*," by Dr. Wm. Murrell, along with which is an article on "Toxins and Anti-Toxins," by Dr. Wm. Murrell and Dr. Joseph McFarland, is of the highest value. The most recent drugs are fully discussed; and to the practitioner in the tropics the use of quinine in enteric fever and the use of tannigen in intestinal flux, together with the whole subject of toxins and anti-toxins, will prove interesting reading.

Part II., "The Dictionary of *Medicine* and *Surgery*," has several articles of special value. The subject of arsenical poisoning, in view of the resemblance of the symptoms induced to those of beri-beri, will be read by practitioners in the tropics who are acquainted with beri-beri with close attention. Tropical diseases are dealt with by Mr. Cantlie. An excellent article on "Vision," by Mr. A. St. Clair Buxton, deals with "errors of refraction and accommodation." The article is written to help those who possibly have little opportunity of rendering themselves familiar with the practical side of refraction, and the writer gives a clear and simple exposition of the subject—just what is wanted by medical practitioners.

Part III. deals principally with sanitation, and the closing chapter, entitled "A Review of New Inventions and Pharmaceutical and Dietetic Articles," is of great practical value.

The Medical Annual for 1902 must form a part of every practitioner's library if he is to keep himself informed concerning the recent advances in medicine and surgery.

Current Literature.

A PHASE IN THE HISTORY OF CHOLERA IN INDIA. BY ANDREW DUNCAN, M.D., B.S.(Lond.), F.R.C.S., M.R.C.P.

Physician to Seamen's Hospital Society; Joint Lecturer on Tropical Medicine, London School of Tropical Medicine; Physician to Westminster General Dispensary.

(Continued from p. 79.)

(9) and (10) THE "IPSE DIXIT" AND EXPERIENCE OF INDIA ARGUMENTS.—These two arguments may be examined together. In his last brochure on cholera, published in 1884, the Sanitary Commissioner with the Government of India claims to set forth "a few

of the great facts showing what has been the experience of India as regards the disease." In the work in question the experience of India is then claimed as showing that human intercourse is never connected with the diffusion of cholera. But is this the experience of India? India as a whole is made up of the Madras and Bombay Presidencies, and of the Bengal Presidency, comprising Assam, Bengal, N.W.P., and the Punjab. Now for the facts. Dr. Cornish, the Surgeon-General of Madras, declared at the inauguration of the Madras Branch of the British Medical Association, that "he was bound to say there that all his individual experience, extending over thirty years, would lead him to deal with the epidemic form of cholera as favoured in its diffusion under certain circumstances by human intercourse." Surgeon-General M. C. Burnell, his immediate successor, at the annual meeting of the same society, declared that his opinion, after "forty years' experience" of the disease, was that water contaminated with cholera dejecta was the cause of cholera epidemics. Surgeon-General Moore, of the Bombay Presidency, has placed on record not only his belief in human intercourse, but even that the disease can be carried by flies. Surgeon-General Townsend for the Central Provinces and the Punjab, and Surgeon-General Payne for Bengal, and Surgeon-General De Renzy for Assam and the Punjab, have expressed the same view. Surgeon-Major Geoffrey Hall, of the N.W.P., at a debate on the disease at the N.W.P. Branch of the B.M.A., said that he had never met a man who agreed with the views of the Sanitary Commissioner for 1879. Surgeon-Major Deakin, of the N.W.P. held the same opinion. Surgeon-General T. Murray addressed an inquiry to 481 medical officers as to the transference of the disease by direct intercourse; 363 answered in the affirmative. Again, Surgeon-General Irvine for Assam believed in human intercourse. Well, we have thus shown that the opinion for Madras, Bombay, the Central Provinces, Assam, Bengal, N.W.P., and the Punjab is in favour of propagation by human intercourse. There does not remain, in fact, much of India left. The above testimony means that the testimony of the above various surgeons-general in its totality is the testimony of the whole of Hindostan, a testimony gathered in most cases from a lifetime spent in the country, and finally a testimony totally opposed to what was declared by the Sanitary Commissioner with the Government of India in 1884 to be "experience of India" at the time.

(11) THE NON-SPREAD BY WATER ARGUMENT.—In the Ninth Annual Report of the Sanitary Commissioner with the Government of India, the spread of cholera by means of water is declared to be a mistake. As an answer to this, I will now relate the history of cholera as it is shown by Dehra Doon, the last station at which I was located in India. Cholera appeared in Dehra Doon in 1887, 1890, 1892, and 1896. Dehra Doon lies at the foot of the Himalayas. Nearest the hill station of Mussooree are the lines of the 2nd Battalion, 2nd (P.W.O.) Goorkha Rifles; then comes those of the 1st Battalion; and, lastly, the regimental hospital. In 1887 the water supply for the regiment was taken

from the Tons stream, above the source of which are Mussooree and Jharipani, and the hill slopes below them. All the accumulated filth on these slopes is washed into the Tons on the occurrence of the first heavy downfall of rain. Numerous filthy villages are on the banks of the Tons, also many villages on the canal from which the water supply is taken off.

In 1887 cholera was present in the villages. The first heavy rainfall occurred on June 28th; on July 1st epidemic cholera broke out in both battalions severely and almost simultaneously.

In 1890 a most excellent new water supply had been delivered to the 2nd Battalion, taken off from a copious natural spring. From the pucca reservoir it was led by pipes into the lines. There is no village above the source of the water. The water supply of the 1st Battalion, of No. 8 Mountain Battery, which had been added to the garrison, and to the hospital, remained as before. In 1890 cholera was again prevalent in the villages. The first heavy rainfall occurred on June 29th. Cholera broke out epidemically on July 2nd in the first Battalion, the Mountain Battery, and the hospital. No cases occurred in the 2nd Battalion lines.

In 1892 the new water supply had been extended to the 1st Battalion lines and the Mountain Battery, but not to the hospital. Cholera broke out at Hurdwar on March 9th, and after some of the pilgrims had returned from Dehra, there occurred a severe epidemic in the city and nearly all the villages in the Dehra Doon valley. This epidemic is said to have been the worst ever known in the Doon. There was now no epidemic in either the 1st or 2nd Battalions; but cases occurred in connection with the hospital, which had not yet the new water supply. There was also no epidemic in the Mountain Battery.

In 1896 cholera was again virulent in Dehra about July 12th. The new water supply was now extended to the hospital. There was no epidemic in either the two battalions nor the hospital.

I have thus submitted this climatic theory to the fullest criticism, and it is found wanting. What else could be expected of a theory that was described by the late Professor Parkes, of Netley, as "revolutionary," and by his successor, Professor De Chaumont, as "Nihilistic"?

In conclusion, we have now seen how the Sanitary Commissioner with the Government of India for 1879 differed from the Sanitary Commissioner with the Government of India for 1867: on every point exactly opposite conclusions are drawn. Now it may be argued that the initial circumstances of place or of mode of dispersal of the pilgrims were different in 1867 and 1879, otherwise how can such diametrically opposite views of the two occurrences be explained? But this argument will not hold water, for the Sanitary Commissioner in 1879 expressly declared that the attendant circumstances were nearly exactly the same as in 1867. Such, then, being the case, it will be, to say the least, surprising to learn that the Sanitary Commissioner with the Government of India for 1867 is one and the same identical officer as the Sanitary Commissioner with the Government of India for 1879. Lastly, inasmuch as the views of the officer

for 1879 had changed from those he held in 1867, it might have been supposed that any one holding the more general view of Indian sanitarians would meet with a certain amount of tolerance. Indeed, in his brochure, "Cholera: what can the State do to prevent it?" the Sanitary Commissioner on p. 106 thus writes: "It has been asserted that medical officers in India have been discouraged and indeed prohibited from reporting facts which favour the contagion theory of cholera. A more groundless assertion was never made." But we have already related the Berar order. And in 1882, Surgeon-General Burnell, of Madras, an officer of thirty-six years' service, when as Sanitary Commissioner of Madras he transmitted his Annual Report to the Sanitary Commissioner with the Government of India, embracing convincing evidence of the propagation of the disease by the pilgrims dispersing from Tirupati, was severely animadverted on for such an expression of opinion. How can such a change of opinion be accounted for? The late Professor Maclean, the Professor of Military Medicine at Netley, on p. 231 of his work on the "Diseases of Tropical Climates," thus writes concerning this change of opinion: "It is impossible to resist the conclusion that, consciously or unconsciously, political considerations weighed with this able officer when he changed his opinion in this remarkable manner. It is certain that the Government of India were alarmed lest, if it could be established that cholera followed the great lines of human intercourse, foreign nations, in their jealousy of British commercial prosperity, should establish, to the detriment of Indian trade, quarantine regulations of an oppressive character. It is certain that Dr. Cunningham and some of the high officials of the Civil Government lost no opportunity in expressing in strong terms their opinion that to reason from facts before them, showing the influence of human intercourse or water on propagating cholera, was highly culpable, and a practice to be reprobated. Any other theory than the one advocated in the Annual Reports of the Sanitary Commissioner with the Government of India was wrong, and the officer who sinned in this way was branded as a mischievous theorist."

With these plain words of my former distinguished teacher, whose eloquent lectures portrayed in stirring language the intricate nature of tropical disease, and whose generous and warm-hearted pen was always at the service of his brother officers, I conclude this account of the theory of cholera as it was formerly ordered in India by Surgeon-General J. M. Cunningham, C.S.I., Sanitary Commissioner with the Government of India.

PLAGUE.

PREVALENCE OF THE DISEASE.

INDIA.—During the two weeks ending February 8th and 15th, the deaths from plague throughout all India numbered 15,165 and 12,675 respectively.

Plague in the Bombay Presidency during February, 1902.—Bombay City, reported plague cases 3,245, deaths 2,561. Bombay Districts:—Panch Mahals, 13 cases, 10 deaths; Kaira, 1,747 cases, 1,088 deaths;

Broach, 809 cases, 607 deaths; Surat, 385 cases, 286 deaths; Thana, 198 cases, 161 deaths; Khandesh, 3,094 cases, 2,324 deaths; Nasik, 287 cases, 231 deaths; Poona, 692 cases, 514 deaths; Satara, 4,846 cases, 3,397 deaths; Sholapur, 1,026 cases, 775 deaths; Ahmednagar, 7 cases, 5 deaths; Kolaba, 121 cases, 101 deaths; Ratnagiri, 69 cases, 51 deaths; Belgaum, 3,125 cases, 2,428 deaths; Dharwar, 3,582 cases, 2,610 deaths; Bijapur, 12 cases, 7 deaths; Kanara, 28 cases, 19 deaths. States:—Hyderabad, 169 cases, 121 deaths; Shikarpur, 5 cases, 2 deaths; Kathiawar, 96 cases, 68 deaths; Cutch, 208 cases, 182 deaths; Rewa Kantha, 231 cases, 158 deaths; Kolhapur, 2,519 cases, 1,632 deaths; Jamjira, 155 cases, 112 deaths; Aundh, 76 cases, 43 deaths; Savanur, 127 cases, 105 deaths; Sachin, 57 cases, 51 deaths; Baroda, 830 cases, 545 deaths. Poona City, 495 cases, 474 deaths; Karachi City, 221 cases, 193 deaths. Europeans, 3 cases, 4 deaths.

EGYPT.—The Director-General, Sanitary Department, reports that in Egypt during the two weeks ending February 16th and 23rd, the number of plague cases were 8 in each week, and that the deaths from the disease were 8 and 5 respectively. The disease prevailed chiefly at Tantah, but isolated cases were reported from Abusir, Mit Ghamr and the neighbouring village of Kom-el-Nur, Alexandria, Mehallet-Abu-Ali, and Zifteh. Since the commencement of the present outbreak to March 8th, 329 fresh cases of plague have been reported in Egypt and 196 deaths from the disease.

CAPE OF GOOD HOPE.—On February 1st, 7 cases of plague remained in hospital in different parts of Cape Colony. At that date plague appeared to be dying out, as but one case of plague (Mossel Bay) was reported at the Cape during the week ending February 1st. There were no deaths from the disease during the week. During the week ending February 8th there was again no fresh case of plague reported, and but one death from the disease—a coloured male—at Mossel Bay. On February 8th four persons, namely, three European males and one male native, remained under treatment in hospitals at Port Elizabeth (1), Cape Peninsula (1), and Mossel Bay (2). The total cases to February 8th in the Cape of Good Hope amounted to 876, of which 638 were males and 238 females; of this number 221 were Europeans. Of the total number attacked 421, or 48.1 per cent., died. The death-rate amongst Europeans attacked amounted to 33.9 per cent.; amongst coloured persons to 56.7 per cent.; and amongst natives to 44.4 per cent.

MAURITIUS.—During the two weeks ending February 27th and March 6th, the number of fresh cases of plague in Mauritius amounted to 7 and 19 respectively, and the deaths from the disease during the same periods to 3 and 5.

SYDNEY.—Plague has recurred in Sydney, and up to March 4th, 46 cases had occurred, and 14 deaths from the disease.

ACTION OF ANTI-PERIODIC DRUGS ON THE PARASITE OF MALARIA.—Lo Monaco and Panichi announce that a drop of a solution of quinine added to a fresh speci-

men of malarial blood, affects the parasites in proportion to its concentration. A weak solution causes them to contract for a few minutes and then expand with marked pseudopodia. This condition of excitement is still more pronounced if a drop of a medium solution of quinine is added to the specimen, and it terminates in the escape of the parasite from the blood corpuscle. A still stronger solution permanently shrivels the parasite and it does not leave the corpuscle. They, therefore, administer quinine in malaria in the dose and solution equivalent to that which produces the detachment of the parasite from the corpuscle *in vitro*—the second phase of the action of quinine. They state that the smallest forms of the parasite are the most resistant to quinine, and that all the parasites grow more resistant the longer the interval since the febrile attack. They conclude from the latter fact that there must be some substance in the blood at the time of the attack which has an antiparasitic action, and the effect of quinine administered at this time is reinforced by this substance already in the blood. This antiparasitic substance affects all the forms of malaria and all species of the malarial parasite with the sole exception of the young unpigmented variety found in æstival tertian. The pigmented forms, on the other hand, are resistant to the action of quinine during apyrexia, but become less resistant during a febrile attack and can then be detached from the red corpuscles with a comparatively weak solution of quinine. But when the malaria has assumed the pernicious form, both the pigmented and the non-pigmented forms display great resistance, probably owing to the absence of the antiparasitic substance in the blood under these conditions, or the presence of some yet unknown antagonistic substance. The quinine behaves the same whether in a solution of distilled water or in a .38 per cent. saline solution. A stronger saline solution prevents these phenomena.—*Riforma Medica Rome*, January 2nd, 3rd, 4th, and 7th, from *Journ. A. M. Assoc.*

CIRCULAR ON THE PREVENTION OF PLAGUE.

(Circular issued in Glasgow, Liverpool, Bristol, &c., modified to suit local requirements.)

THE DESTRUCTION OF RATS.

To Owners and Occupiers, Warehousemen, and Others.

WHEREAS it is expedient that rats in every English city should be destroyed, the Health Committee invite the careful attention of owners and occupiers, warehousemen, and others, to the following memorandum, and their co-operation in the measures suggested, which, for the greatest safety, would be best carried out before any actual invasion occurs. Participation in this work should be accepted by every citizen as part of his municipal duty at the present time.

In the event of the introduction of plague into the city, its extension would be best met:—

- (1) By wholesale destruction of rats.
- (2) By their exclusion from dwellings, warehouses, and places of business generally, and by the destruc-

tion of their haunts and feeding-places in the vicinity of dwellings.

To be effective, the effort must be general, and strict watch should be kept for the appearance of rats in places not at present infested by them.

Rats gain access to buildings chiefly:—

(1) By burrowing through the earth below wooden flooring.

(2) Along the course of drainage and other pipes led through main walls in holes which are too large, and up the course of rain water pipes.

(3) By the badly-fitting doors, doors broken at the foot, and other openings on ground-floors.

This is to be met:—

(1) By cementing or asphaltting earthen basements when these are burrowed.

(2) By packing loosely-fitting holes through which drain or other pipes pass.

(3) By refitting doorways, protecting the foot if necessary with sheet-iron, and by protecting openings in basements by wire netting if nothing better can be done.

Rat-runs in buildings should be discovered and destroyed.

It is useless to stop rat-holes in a house and leave the means of access to the tenement still open. Rats overrun a building behind the plaster and woodwork, in the casing of pipes, and below flooring, but they gain access at the basement.

Rat-infested premises may be dangerous to the health of the tenants and a menace to the neighbours, and may thus be dealt with under the Public Health Acts.

Landlords are requested at once to inquire into the presence of rats on their properties, and to take action on the above or on similar lines.

Rats are attracted to buildings in search of food.

Ashpits, collections of garbage, stable yards, &c., afford them food; lumber-heaps, or disused structures, are suitable for nesting.

Owners are reminded that ashpits that are allowed to be a nuisance may be dealt with under the Public Health Acts.

Slaughtermen and others are warned that contraventions of, and neglect to observe, the bye-laws with regard to the removal of garbage are punishable.

All household refuse should, as far as possible, be burned by the householder.

The Health Committee rely on the willing co-operation of landlords and householders in these directions, and tenants will forward the work of repression by informing them of premises which are rat-infested.

TO DESTROY RATS.

Trapping and poisoning are the most readily available methods; hunting with dogs, ferrets, or a mon-goose is sometimes practicable, but they can rarely follow the rat into its burrow.

Trapping.—The most useful form of trap is a spring trap, baited with ham. Cage traps are too large, and the rat soon learns to avoid them.

Poisoning.—Some form of arsenic or phosphorus paste is to be preferred. There is no available poison which will preserve the dead rat from putrefaction and prevent smell.

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Archives Russes de Pathologie, de Médec. Clinique et de Bacteriologie.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Brooklyn Medical Journal.
Caducée.
Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Esercito
Hong Kong Telegraph.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
Journal of the American Medical Association.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Medical Record.
Medical Review.
Merck's Archives.
New York Medical Journal.
New York Post-Graduate.
Pacific Medical Journal.
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- 4.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.
- 5.—Correspondents should look for replies under the heading "Answers to Correspondents."

The Journal of Tropical Medicine.

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Original Communications.

PRINCIPLES DETERMINING THE GEOGRAPHICAL DISTRIBUTION OF DISEASE.

By LOUIS W. SAMBON, M.D.(Naples).

Lecturer to the London School of Tropical Medicine.

(Continued from p. 95.)

WHILE each parasite has its special limit of host species, each species of plant or animal has its particular parasitic flora and fauna. If we take the black poplar as an example, and count all the plants and animals which live upon it, within it, or in association with it, we shall find that they number not less than fifty. Its roots are covered by the mycelia of a fungus, and pierced by the suckers of toothwort plants. The toothwort withdraws the juices absorbed by the roots through the instrumentality of the symbiotic fungus. Meanwhile, in the cavities in the leaves of the toothwort, various small animals are caught and made use of as nitrogenous food. Again, the poplar tree bears mistletoe on its boughs, and its presence is due to the missel-thrush. The thrush takes the mistletoe berries for food, and in return, renders the plant the service of dispersing the seeds and establishing them on other trees. The parasitic mistletoe takes its liquid nutriment from the wood of the poplar tree; but, on the other hand, its own stems are covered with lichens and these lichens are themselves a symbiotic community of algæ and fungi. Within the wood of the poplar stem spread the mycelia of certain basidiomycetes (*Panus conchatus* and *Polyporus populinus*), whilst the leaves are covered with a little orange-coloured fungus, *Melampsora populina*. In addition, no less than three gall-creating species of pemphigus live on the leaves and branches of the poplar, and a number of beetles and butterflies are nourished by them. Certain lichens, mosses, and liverworts regularly settle on the bark of

old trunks, and included amongst these may be the species of liverwort which is inhabited by rotifers.

In some cases (*Monogenea*) the complete evolution of a parasite only requires one host, in other cases (*Digenea*) it demands two, successive and, in general, specifically different hosts. Thus, whilst *Hymenolepis murina* goes through its entire life-cycle without any change of host, *Taenia saginata* spends its larval stage within the tissues of herbivorous mammals, and its adult stage within the intestine of certain carnivora. The two necessary hosts may belong to widely-sundered groups of the animal kingdom. *Dipylidium caninum* spends its adult stage in a mammal (cat or dog) and its larval stage in an insect (*Thichodectes canis* or *Pulex serraticeps*).

The distribution of parasites requiring two hosts, belonging to two different groups is, of course, limited to those areas in which both hosts are simultaneously found. However, the geographical distribution of heterogeneous parasites is often very complex, because, although only two hosts are necessary for the complete evolution of their life-cycle, one or both hosts may be represented by a number of species.

The adult *Fasciola hepatica*, which causes the disease called "liver rot" in sheep, is found in a large number (about twenty-five) of domesticated and wild animals, and its *redie* develop in several species of fresh-water snails (*Limnaea*). Thus, the number and sub-cosmopolitanism of both its definite and intermediate hosts fully explain the wide geographical distribution of this trematode.

The definitive host of our malarial parasites is represented by several species of *Anopheles*, but the geographical distribution of the intermittent fevers does not coincide exactly with the geographical range of these insects. *Anopheles maculipennis* is found in several parts of Italy in which malaria is unknown, and it is still quite plentiful in England in all those places in which ague was once very prevalent. So far, no other animals have been found to foster the endogenous cycle of our malarial parasites; but such

a possibility is not altogether excluded, and the three species of hæmocytozoa, found by Dionisi in bats, are strikingly like our own parasites. It is not absolutely unreasonable to surmise that the disappearance of ague in England may be connected with the extinction of some animal, or possibly some plant, which may have been a necessary link in the chain of its natural history. As an example of a species which has lately become extinct within the ague districts of England, I may mention the *Large Copper Butterfly*, which was formerly found in abundance in the Cambridgeshire fens and nowhere else in the world.

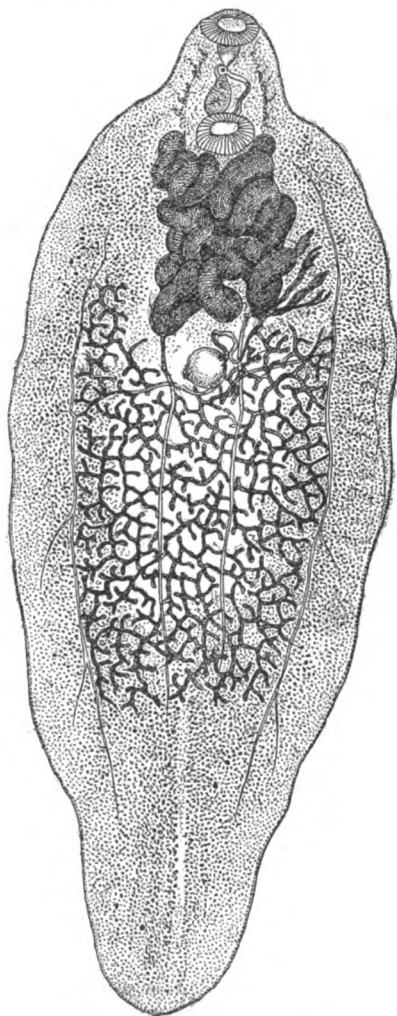


FIG. 13.—*Fasciola hepatica*.

Even after the establishment of the parasitic theory in the causation of disease, it would have been difficult to account for the geographical distribution of diseases so long as plants and animals were believed to have originated in the areas they now occupy, or climate was considered to be the principal factor in their distribution. Now, we know that the geographical distribution of plants and animals is due to the co-operation of many factors, and that any solution which does not recognise all the factors is bound to be erroneous.

The most important conditions determining the distribution of species are not the meteorological conditions, as was formerly believed, but association and competition with other plants and other animals. When the purely physical conditions were considered paramount, it was difficult to explain why one region should differ so greatly in its floral and faunal aspects from another whose physical characteristics were practically identical with its own, or why certain plant and animal assemblages should enjoy an almost limitless or universal extension, while others, without apparent reason, should be circumscribed within very narrow limits.

To explain the influence of the animate environment in determining distribution, I may mention that red clover would not grow in New Zealand until bumble-bees were introduced to fertilise its flowers. Now, it displaces the native grasses. Darwin points out that cats are also in a measure responsible for the productions of clover seed in England, through the interrelations of cats, field-mice and bumble-bees.



FIG. 14.—*Limnaea truncatula*.

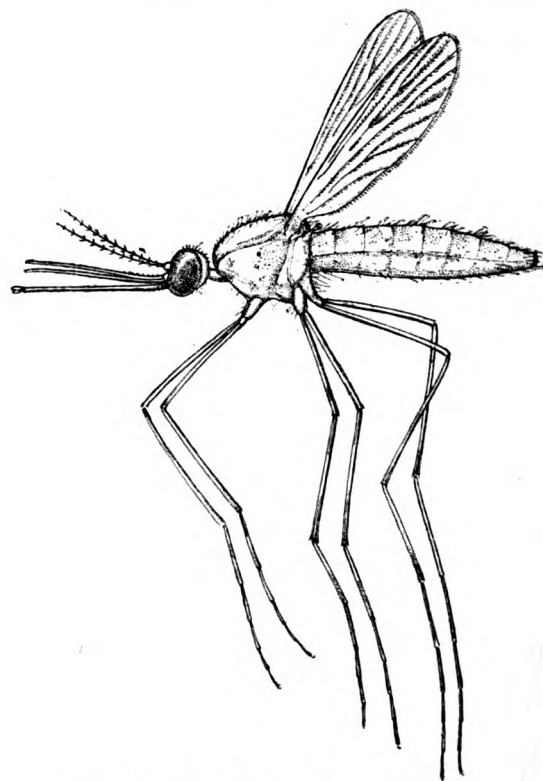


FIG. 15.—*Anopheles maculipennis* (female).

Numerous flowering plants, such as arbutæ, ericaceæ, and rhododendrons, cannot thrive unless the soil contains certain fungi, with which they are invariably associated. The fungus covers their roots with a felt-like mantle of hyphæ which assume the function of root-hairs, and supply the green-leaved

plant with nutrient materials from the ground, while the green-leaved plant supplies the mycelium with substances elaborated above in the sunlight. This association, called *mycorrhiza*, probably originated as a form of parasitism, but has become a true symbiosis similar to that of a lichen thallus. A similar association is that of certain bacteria with many legume plants, such as peas and beans. The bacteria live on the roots in little wart-like outgrowths, called "root-tubercles," and supply the host-plant with nitrogen absorbed from the air circulating in the soil.

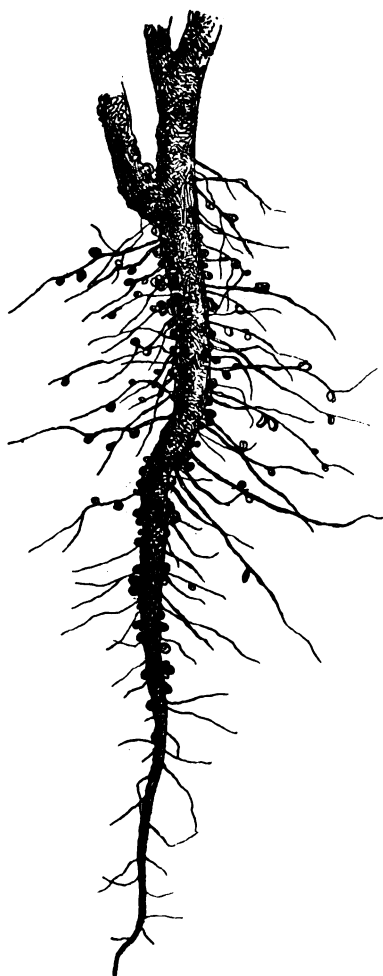


FIG. 16.—Root-tubercles on *Vicia faba*.

The conditions which determine the abundance of a species and the extent of its range are often greatly complicated. Conditions are dependent upon conditions in an almost endless chain. Howard has recently proved the existence of several fatal tertiary parasites and the probable existence of quaternary parasites with *Orgyia leucostigma* in Washington. Upon the condition of this chain of interdependencies rests the welfare of the primary host. If adverse conditions affect the quaternary parasite, the primary host suffers, for the tertiary parasites increase and kill off the secondary parasites, allowing an increase of the primary parasites which kill off the *Orgyia*.

In studying the geographical distribution of diseases, apart from any preconception as to their causation, we find that its salient facts are identical with those of zoo-geography.

Some diseases, like typhoid fever, tuberculosis, pneumonia, and dysentery, have a very wide distribution; others, like endemic hæmaturia and dracunculiasis, are encompassed within narrow limits; others, again, like cholera and plague, while usually occupying a restricted habitat, may, at times, under favourable conditions, spread very widely. This is exactly what we find in the distribution of animals. Some, like the small serotine bat (*Vesperugo serotinus*), the fish-hawk (*Pandion haliaëtus*), and the common red river-worm (*Tubifex rivulorum*), have a world-wide range; others have a very restricted habitat. Several species of humming-birds are restricted respectively to the volcanic peaks of Chimborazo and Pichincha in the equatorial Andes, and to the extinct crater of Chirigui, in the province of Panama, Columbia. Certain fishes are limited to a single lake: thus the Lough Killin charr is confined to the lake of that name. Examples of animals which may occasionally spread out of their usual habitat are the Norwegian lemming and the migrating locust. Too much stress should not, however, be laid upon what would appear to be the absolute localisation of a species, since such supposed localisation is frequently only the expression of defective knowledge. The famous South American oil-bird (*Steatornis caripensis*), for example, was for a long time believed to inhabit solely a cave near Caripé, in the province of Cumana, Venezuela. Now we know that it has a comparatively broad area of distribution which embraces Sarayacu and Caxamarca in Peru, Antioquia in Columbia, and the Island of Trinidad. *Schistosoma hæmatobium*, the parasite which causes endemic hæmaturia, was believed to be restricted to the African continent; more recent research has proved it to have a wider distribution in the adjacent islands, on the Arabian coast of the Red Sea, and in Mesopotamia. Verruga seems to be confined to certain valleys of the Peruvian Andes; but, if like *coko*, *parangi*, and *purru*, it is nothing more nor less than yaws, then, of course, it has a very wide geographical distribution.

The usual method of indicating on maps the distribution of diseases is by colouring the whole area of their geographical range. But although perhaps this may be the only possible method on very small maps, it is greatly misleading, because, even when the distributional area of a disease is continuous, its stations rarely form one continuous tract. A suitable illustration is that of the intermittent fevers. Their distribution is very wide, but each type of fever has its own peculiar distribution, and in the various regions of its habitat it is only found in certain stations where the suitable circumstances occur; usually low, swampy districts, infested by *Anopheles*. To map out correctly the distribution of these fevers, a different colour should be adopted for each fever, and their various stations should be marked by dots, which would thus permit the overlapping of the various fevers in proportion to their prevalence. The map would thus be coloured by series of closely-set but separate variegated patches. In disease distribution

as in zoo-geography we must distinguish between *locality* and *station*. Animals inhabiting forests or moorland, or pools, are only found where such physical conditions occur.

The geographical distribution of disease is subject to variation. Indeed, it is just as unstable as that of animals and plants. Some diseases become extinct, or their prevalence diminishes, and thus endemic areas become restricted; others shift their habitat, spread more widely, become more prevalent, and invade new countries. Leprosy has become almost extinct in Europe; on the other hand, small-pox, scarlet fever, whooping-cough, and the chigoe-pest have spread continuously, invading new countries.

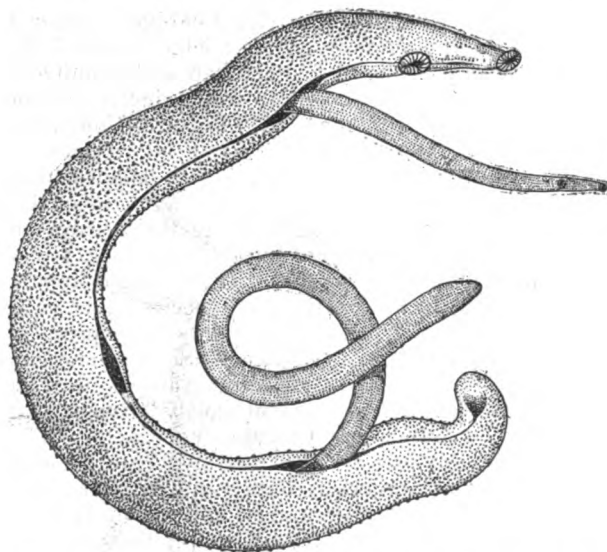


FIG. 17.—*Schistosoma hematobium*. (Female enclosed in gynephoric canal of male.)

The intermittent fevers are a good example of the changes and fluctuations in the distribution and prevalence of certain diseases. These fevers have entirely disappeared from England, where, at one time, they used to be very prevalent; they have invaded new places, such as the Islands of Mauritius and Réunion, where they were previously unknown, and, in Southern Italy, they receded before the practical sanitation of the old Greek colonists, to return once more, like a tide, as soon as the land fell out of cultivation.

These variations are similar to those which take place in the distribution of animals and plants. The bear, the wolf, the wild boar, the beaver, have become extinct in England; the lion has disappeared from Greece; whales have been restricted to the Polar regions. On the other hand, the brown rat, the rabbit, the sparrow, the Ligurian bee, the thistle, have spread very widely.

Some diseases, when first imported into a new country, have at once spread like wildfire; others, although repeatedly imported, have never become endemic in the new locality; others, again, have become acclimatised in some places and not in others.

The *chigoe*, which formerly was confined to Central America and the West Indies, was carried in ballast

in 1872 on a vessel from Rio Janeiro to the coast of Guinea. Thence it spread over the greater part of Africa with incredible rapidity. On the other hand, *Filaria loa*, though frequently introduced by Negro slaves into the West Indies, has never become acclimatised in those islands. All these peculiarities in the geographical distribution of disease, which could not possibly be explained by any of the old theories, find at once a satisfactory explanation in the parasitic theory. The capacity of a species for transportation and acclimatisation in a new environment depends upon the degree of simplicity of its ecological conditions. These conditions are still very imperfectly known. Thus, taking an example from the Diptera, we do not know why *Calliphora erythrocephala*, *Cyrtoneura stabulans* and *Stomoxys calcitrans* should have been introduced at an early date and flourished to excess in America and many other countries, while *Sarcophaga carnaria* is unknown in any of them.

Very many species are constantly being introduced, yet, failing certain conditions, such as an appropriate season at the time of importation, they may not establish themselves for centuries in places which seem to offer the most suitable environmental conditions.

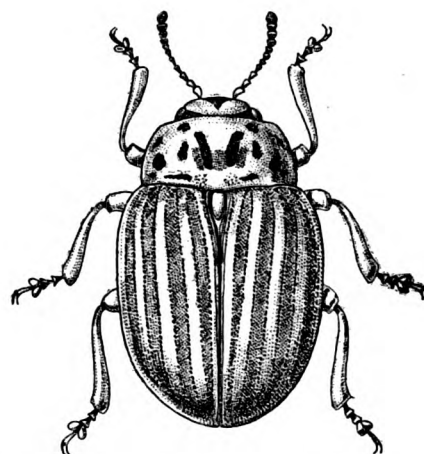


FIG. 18.—Potato-beetle (*Chrysomela decemlineata*).

An interesting example with regard to the distribution and prevalence of disease is that of the rôle played by *Solanum rostratum* in connection with the ravages of the potato-beetle (*Chrysomela decemlineata*). *Solanum rostratum* is an undesirable weed or thistle, on account of its prickles. It is a subglobose plant, and has the tumble-weed habit, i.e., when ripe, it snaps off close to the ground and goes bowling along before the wind at a great rate; but it may also travel by means of animals, its prickly seed-pods becoming entangled on the tails of cattle. It was noticed in various parts of America where the Colorado beetle had spread that its ravages diminished with the advent of *Solanum rostratum*, and augmented whenever this weed was eradicated. The cause is that *Solanum rostratum* is the native food-plant of this beetle, and that only when it is scarce the nearly related potato-plant (*Solanum tuberosum*) is accepted as a substitute.

The part played by plants and animals in the propagation of disease-parasites is becoming more evident every day. In 1877, Dr. Manson proved that mosquitoes were the propagators of filarial diseases, because he discovered that *Culex pipiens* was the intermediary hosts of *Filaria bancrofti*. In 1898, Ross proved that certain mosquitoes of the genus *Anopheles* were the definite hosts and disseminators of malarial parasites. It has long been known that flies can carry about the pathogenic parasites of plants and animals, just as they carry the fertilising pollen of flowers on the hairs of their claws, and now the rôle of *Musca domestica* in the propagation of anthrax, cholera, ophthalmia, and typhoid fever is fully recognised. The association of *Trichina spiralis* with hogs and rats; of *Bacillus pestis* with rats,

The cause of epidemics seemed at one time an inscrutable mystery. The physicians of some decades ago spoke of a *genus epidemicus*, and Noah Webster, the great lexicographer, who was a physician, strove to establish a connection as cause and effect between earthquakes, hurricanes, volcanic eruptions and the outbreak of great epidemics. Now, in the light of the parasitic theory, the cause of epidemics is quite obvious. Disease epidemics occur exactly in the same way, and for the same reasons, as animal pests, that is to say, they are due to an excessive increase of the pathogenic agent under very favourable conditions. Field voles in "vole years" appear in our fields in enormous numbers, and literally honeycomb the soil. Unable to explain their prodigious increase, the ancients believed that they occasionally rained down from the clouds. During damp autumns and late summers, field-snails appear as if by magic in wondrous numbers, and may bring about serious epizootics of tape-worms amongst fowls.



FIG. 19.—Foot of house-fly, with spores of various parasitic fungi.



FIG. 20.—Wing of grass-fly, with spores or conidia of the potato fungus (*Peronospora infestans*).

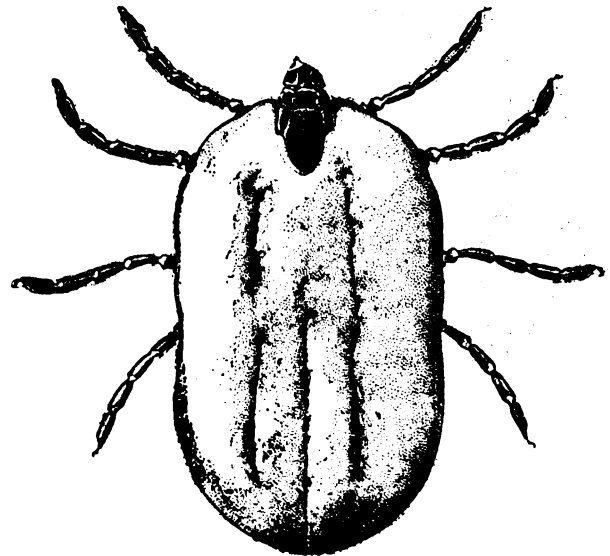


FIG. 21.—*Ripicephalus annulatus* (female).

marmots and fleas; of *Tenia echinococcus* with sheep and dogs; of *Dyplidium caninum* with *Pulex serraticeps* and *Tricodectes canis*; and of Nagana disease (*Trypanosoma brucei*) with the tsetse fly (*Glossina morsitans*), are well-known examples.

A very interesting association is that of the cattle-tick *Ripicephalus annulatus*, with Texas fever. This tick does not carry the disease in a direct manner from a diseased animal to a healthy one, because it does not usually change host as was at one time believed, but the female tick transmits the protozoal parasite (*Piroplasma bigeminum*) of Texas fever to its progeny, through the egg, and the young larvae carry it to the cattle on which they in turn become attached, and probably only the female ticks inoculate it when, at maturity, they imbibe blood.

No doubt we derive the largest proportion of our disease from those animals with which we come into daily contact, such as cattle, house pets, city insects and household pests, and above all from those which are used as food.

Any species whatsoever, if not restrained by adverse conditions, would soon increase to such an extent as to fill the whole world with its progeny. Crampe has clearly proved that the fabulous multiplication of field-voles, under favourable conditions, finds its explanation in the great fertility of the females, in the appearance of several successive generations in the same year, and in the numerical preponderance of the females over the males.

The fertility of some species is really wondrous. A queen bee lays about five million eggs in her lifetime of four or five years. A female white ant produces eighty thousand eggs a day steadily for several months.

The greater the danger to which an animal is exposed as a result of structure, mode of life, or development, the greater are its powers of increase. Animal parasites which pass from one host to another, or which spend a part of their existence in a free state, run such a risk of not finding a suitable host, that they are necessarily endowed with enormous powers

of multiplication. But, however great the increase of certain species, the crowd of life is such that there is little danger of one species flourishing to the exclusion of all others.

Although the appearance of most pests is due to local increase, some may be due to migration. The Scandinavian lemming and the devastating locust are well-known examples of animals which migrate occasionally on account of unchecked multiplication and scarcity of food in the region which they inhabit.

The migrations of the lemming occur at intervals varying from five to twenty years. These little arctic mammals move as an army, steadily and slowly, advancing always in the same direction and regardless of all obstacles, swimming across streams, and even lakes of several miles breadth. The migration lasts from one to three years, and usually ends in the total destruction of the migrating swarms.

The migrations of the Rocky Mountain locust (*Caloptenus spirtus*), have been thoroughly investigated by the United States Entomological Commission. The history of the American locust is in nearly all respects parallel with that of the locust of the old world. Both insects have a permanent breeding area, and both, periodically, under special favouring conditions, multiply in enormous numbers, and migrate far beyond their usual habitats.

The permanent breeding region of the Rocky Mountain locust is approximately 300,000 square miles, and lies mainly between longitude 102° and 114° west of Greenwich and latitude 63° and 40° north. The locust does not breed continuously over the whole extent of this area each year; but while for a series of years it may deposit its eggs in a given river valley, in some park, or in some favourable area on the plains lying about the mountains, in a certain year or for several years in succession, it may desert its customary breeding grounds for adjoining regions, or cross a low range of mountains and breed in a more distant valley. Moreover, the true breeding grounds in this area are for the most part confined to the river bottoms, or sunny slopes of uplands, or the subalpine grassy areas amongst the mountains, rather than continuously over the more elevated, dry, bleak plains. In vertical distribution it may be said to breed from an altitude of about 2,000 feet up as far as 10,000 feet, or near the timber line in the Rocky Mountains, though few probably breed in great numbers above an altitude of 8,000 feet.

When the locust multiplies in great numbers it is liable to spread out of its permanent breeding ground, and invade a wider area which extends over the elevated plains east of the Rocky Mountains and includes a large portion of British America. This region is known as the *subpermanent region*, but there are no natural barriers between the permanent and subpermanent regions, one region shading imperceptibly into the other.

In certain years the multiplication is excessive, and then the migrating swarms push further east into what has been called the *temporary region*. The limit of this region is the Mississippi, which, as Mr. Walsh first pointed out, the locust never crosses.

The bearing of these facts on the study of epidemics is quite apparent.

Many of our most formidable diseases have been imported, like noxious weeds and injurious insects, from one country to another through the agency of man. The opening up of the vast tropical belt, the wondrous growth of international trade, the greater rapidity of transit, and the safer carriage of goods of all kinds, have enormously increased the opportunities of accidental introduction of foreign diseases. There are several diseases potentially cosmopolitan which threaten to invade new countries, amongst such are beri-beri, blackwater fever, yaws, and sleeping-sickness. A restricted habitat is no evidence of inability to spread. The *Coccidæ* under natural conditions, have usually a rather restricted distribution, but by means of commercial distribution of nursery stock many of them have become of almost world-wide range. Fortunately their enclosed hymenopterous parasites have spread almost equally with their hosts. We must therefore prepare to prevent the spread of exotic diseases by appropriate legislative measures, such as those adopted by New Zealand, Australia, Cape Colony, and the various American States to protect themselves from the introduction of insect pests and dangerous weeds. But to be able to lay down exact prophylactic rules, we must be fully acquainted with the life-history, ecology and present geographical distribution of the pathogenic agents we wish to avoid.

In the domain of agriculture man has already achieved wondrous success. The locust plagues of North America have been almost stamped out, and Riley saved the orange orchard of California by pitting the *Coccinellidæ* against the *Coccidæ*. Similar results can most certainly be obtained in human pathology, and, indeed, recent experiments on the prophylaxis of malaria and yellow fever have proved most successful. Last year, the Sanitary Department of the U.S. army succeeded in eradicating yellow fever from Havana where it had been constant for the last 150 years, simply by putting into act and fact the recently acquired knowledge that the *Stegomyia* mosquito is the carrier of yellow fever and that this fatal disease is not transmitted in any other way.

NOTE ON THE SPREAD OF YELLOW FEVER.

By C. CHRISTY, M.B., C.M.Edin.

Thompson Yates Laboratory, University College, Liverpool.

In reading through a series of most interesting papers published in the *British Medical Journal* during the years 1841-42, by E. J. Burton, M.D., on the diseases he met with during his term of service at Sierra Leone, I find a footnote (p. 309) of peculiar interest at the present date in the light of recent revelations in the etiology of yellow fever and for other reasons. Dr. Burton writes:—

"It being at present [that is, 1842] almost generally supposed that yellow fever is neither contagious nor propagated by infection, I wish particularly to mention the grounds on which it is stated that this disease was introduced and propagated by contagion amongst the inhabitants of the Island of St. Mary's, in the year

1837. The crew of Her Majesty's brig "Curlew," I think, but I cannot be quite positive as to the name, contracted the yellow fever at Sierra Leone in 1837, where it was then raging. She sailed from that place for change of air, and entered the port of St. Mary's, having at the time some of her officers and crew labouring under the disease. At the period of her arrival St. Mary's was quite healthy and free from *fever of any kind*. Some of the officers afflicted with yellow fever were landed and brought to the house of the Colonial Surgeon, Mr. Tebbs, who attended them; he was, in a few days, seized with the disease and died, although a 'seasoned person.' The fever now spread rapidly, the first cases all being traced to have had communication with Mr. Tebbs or his patients. That this fever was imported into St. Mary's, and then propagated by contagion, is strongly supported by the fact that the disease commenced in this place, *previously healthy*, immediately after the arrival of the vessel having the fever on board, and that it never had before appeared in that settlement, nor has it ever appeared since. It is not wished to maintain that yellow fever is always contagious, or always propagated by the same cause, but the fact of its having been imported and propagated in this instance by contagion is given as a simple fact, and not for the purpose of supporting any particular theory or argument."

Burton apparently had very good grounds for believing this to be a clear case of contagion, but reading between the lines, we now realise that it was a very pretty example of indirect infection. The Island of St. Mary is off the north-west coast of Sierra Leone.

WATER ITCH, OR SORE FEET OF COOLIES.

By WILLIAM E. LLOYD ELLIOTT, M.D.

Late Medical Officer of the Assam Frontier Tea Co., Dibrugarh, Assam.

In a paper by Dr. Dalgetty (Sylhet), in the JOURNAL OF TROPICAL MEDICINE on March 1st, 1901, on water itch, he comes to the conclusion that the disease is caused by an acarus, of which he gives a minute and interesting description. In this article Dr. Dalgetty alludes to an article published by me in the JOURNAL OF TROPICAL MEDICINE, December 15th, 1900.

I think, however, that Dr. Dalgetty's conclusions and methods of investigation are open to various objections.

The presence under the microscope of "the chief organisms of putrefaction," "rapidly moving diplobacilli," "faecal matters," "pus cells," "particles of sand," "fibres of cloth and other extraneous matters," shows that he must have examined the vesicles in the pustular stage, and in many cases after they had been contaminated by rupture, as all these things could hardly have passed through the unbroken skin. I think the method should be to examine in the early vesicular stage before contamination can possibly have taken place, or, as suggested by Dr. Bentley (*British Medical Journal*, January 25th, 1902), by removing portions of the skin with vesicles intact, and making sections with a microtome.

The presence of the acarus in the crusts found on sores, or in crusts, &c., exposed to the air on a slide for several days, is surely no proof of the acarus having caused the disease, the crusts having been in both cases open to infection from outside sources.

I do not agree with Dr. Dalgetty that the disease "spreads" in any true sense, as it would if the acarus extended its burrow, for I have never known it to do so. The vesicles may come in successive crops, but the new ones are discrete. The only variety (the Herpetiform) which looks at all like an extended burrow, takes that form from the first, and is not formed from the centre outwards.

NOTE.—My original article was inserted anonymously owing to a misunderstanding.—W. E. (see page 112 of this issue).

"SURRA," OR EQUINE RELAPSING FEVER.—The presence of this disease in the Philippines was discovered October 16th, 1901, by Captain Allen Smith, Assistant Surgeon, U.S. Army, Surgeon in charge of the Army Pathological Laboratory, and Dr. J. J. Kinyoun, surgeon Marine Hospital Service. Smith and Kinyoun, examining the blood of a sick horse, found an actively motile body which, on investigation, proved to be apparently identical with the parasite found by Evans in India and Burma in 1881 in the blood of horses, mules and camels ill with what the natives called "surra," which means "rotten." This parasite, the *trypanosoma Evansi*, occurs in enormous numbers in the blood of animals ill with the disease. This disease has never been recognised, to our knowledge, before in the Philippine Islands. Large numbers of American horses and mules, as well as native ponies, have died during the past six months. In some provinces there are Pueblos without a single pony. In the main corral here in Manila some 200 and more horses and mules have died from a "wasting" disease during the past four months. This disease was generally diagnosed "glanders," and hundreds of animals were killed, until General Chaffee stopped it by a general order. Investigation has progressed to such a point that it is possible to state positively that this fatal epidemic among horses, mules and ponies is *not* glanders but "surra." The blood of nearly a hundred horses and mules, most of whom were diagnosed as having glanders, was examined. But little glanders was found in the Manila corrals; four-fifths of the deaths among Government horses and mules during the past three months have been from "surra." How the disease is disseminated is not known, but it has been induced in healthy animals—horse and monkey—by the subcutaneous injections of the blood of a horse ill with surra whose blood contained the *trypanosoma*. Treatment seems of little avail; quinine and methylene blue do not seem to affect the course of the disease. Work is now being done especially in the direction of the mode of transmission. The evidence points to transmission by some sucking insect.—*Boston Medical and Surgical Journal*, January 30th, 1902.

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THE

Journal of Tropical Medicine

APRIL 1, 1902.

SMALL-POX AND ITS PROPHYLAXIS IN ORIENTAL COUNTRIES.

THE subjects of small-pox, vaccination and re-vaccination are being widely discussed in London and in several of the large cities of Great Britain at the present time.

We think a great deal of information could be given by medical men in the tropics on the subject of small-pox and its prevention, could they be induced to come forward and state their experiences, and the conclusions gathered from their experiences. One observer, Hem Chandra Sen, M.D., Calcutta, of the Campbell Medical School and Hospital at Sealdah, India, ventured, in the *Indian Medical Record* of February 19th, 1902, to formulate a statement upon "Recent vaccination as a preventive measure against both small-pox and plague." In his article he states: "Recent vaccination [for small-pox], as far as my

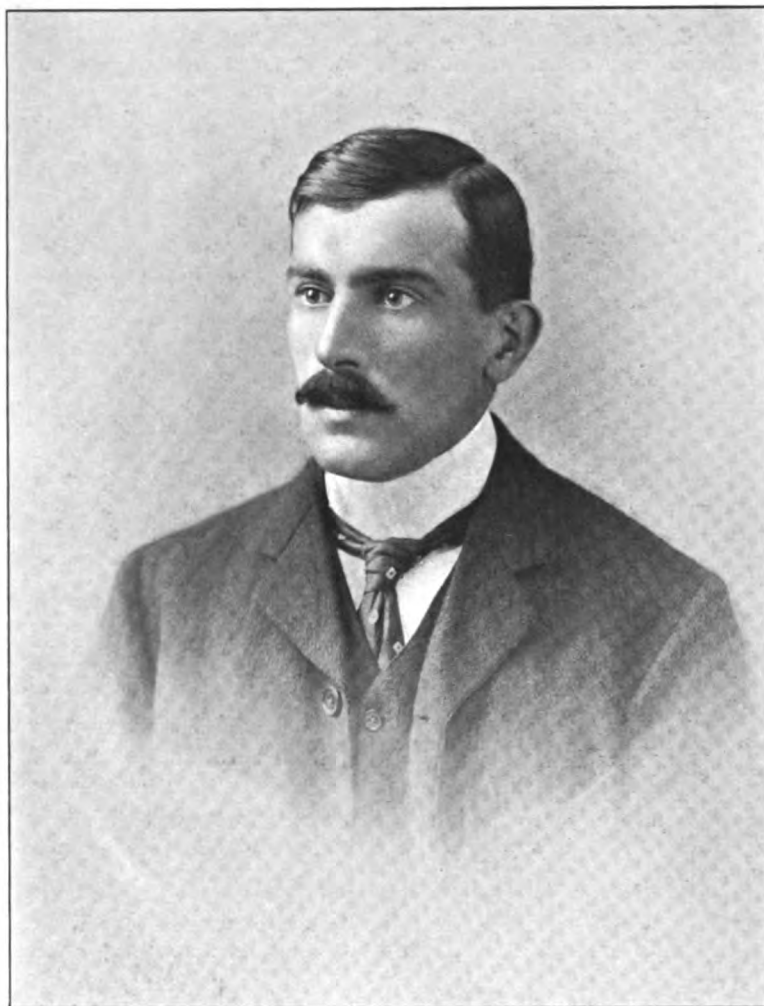
observations are concerned, prevents people from having an attack of plague in a vast majority of cases." Dr. Sen assigns the reason for this immunity from plague in vaccinated people to be due to the fact "that if any disease germ is introduced into the system, the vital force adds to the blood antitoxin to neutralise the introduced poison." He ascribes the protection to be due to increased activity of the phagocytes, and aptly illustrates this activity thus: "When the armed militia, or phagocytes, which guard the living human frame are stimulated with a mild virus like vaccine lymph to be on their guard, no stronger poison can do harm so long as they are properly on the alert."

Concerning the credit thus claimed for vaccination as a protection against plague as well as small-pox it is not our intention to discuss, but Dr. Sen's remarks may elicit opinions from others who are in a position to judge upon this matter.

It is a mistake to think that protection against small-pox is a new subject to the Oriental native. Prophylaxis by inoculation was known and practised in India, in China, and in other countries, before European ways and customs entered into the daily life of the inhabitants of these regions. In fact, it is more than probable that the west learned inoculation from the east.

The Chinese acquired immunity by inoculating in one of three ways. The infected person's clothes were worn by the healthy; or the contents of small-pox pustules were applied to a scratch in the skin, just as we vaccinate at the present day; or a dry small-pox scab was reduced to powder and blown up the nostril.

Vaccination with lymph was not known in China until the year 1801, when vaccine matter was introduced by traders belonging to the old East India Company. In the Philippines vaccination was introduced by the Spaniards about the same date. The difficulties of introducing vaccine from Europe to the Far East in the early days of the nineteenth century, when sailing ships were then the only means of transmission, were such that some intermediate form of lymph production had to be established. The British attained their purpose from India; and the Spaniards, by travel-



THE LATE PATRICK THURBURN MANSON, M.B.Lond.

Who died March, 1902, at Christmas Island, whilst engaged in the scientific investigation of Beri-Beri.

ling across South America and renewing their lymph source on the way, ultimately reached the Philippines across the Pacific.

But vaccination has attained but little hold on any Asiatic people, and revaccination may be said to be, except under compulsion, unknown and unpractised. The adult natives, and especially those who have been vaccinated in their infancy, resent being vaccinated. The parents may consent to have their child done, but in very rare instances will they themselves undergo treatment.

Amongst the Chinese and in the Philippines vaccination to a certain extent has been going on ever since it was first introduced. The arm-to-arm method has been carried on, after a fashion, during the whole of the nineteenth century, but the virus used at the end of the century was the result of the vaccinations made at the beginning of the century, for the lymph was kept alive by arm-to-arm vaccination merely. The result was an expended and exhausted lymph and an imperfect or wholly abortive protection. Vaccine matter, as we know, loses its power as it passes from arm to arm after a certain time—some eight or nine transmissions; but when continued for a century its attenuation must be reduced to the minimum.

Pustules, the result of vaccination by a Chinese "doctor" using the arm-to-arm vaccine matter, are quite small, about the size of a pea, and set up but little local irritation. Consequently the Chinese prefer the milder form of acquiring "protection," holding that the European matter is "too strong for them." They in fact go through the form of vaccination without being in any way protected. With such attenuated lymph it is no wonder that vaccination has fallen into disrepute amongst a keenly observant people like the Chinese.

When reporting therefore upon the protection afforded by vaccination amongst the better-known Oriental people, it is necessary to take note of these facts, and to remember that the native may have gone through the form of vaccination without being in the least protected. That small-pox is rife in the East every one knows, and it behoves all travellers to get revaccinated before

starting from Europe; and all residents in warm climates should be revaccinated every time an epidemic of the disease threatens in their neighbourhood. Sanitation may be the ideal form of protection against small-pox, but the Orient is not yet sanitary; and even the most rabid Anti-vaccinationist, if he means to travel, will be wise to drop his fad for the nonce. Anti-vaccinationists, however, like most other narrow-minded faddists, seldom travel; did they do so their "Leagues," or whatever they style themselves, would soon cease to exist, and their pernicious and damaging doctrines be heard of no more.

MEDICAL OFFICER OF HEALTH FOR BANGKOK.

THE RETIREMENT OF DR. P. A. NIGHTINGALE.

DR. H. CAMPBELL HIGHET has been appointed Medical Officer of Health for Bangkok in place of Dr. Nightingale, resigned. Dr. Highet holds the D.P.H. diploma, and is well qualified to take up the important post vacated by Dr. Nightingale. We have been indebted to Dr. Highet from time to time for excellent articles on tropical ailments which appeared in the JOURNAL OF TROPICAL MEDICINE and other journals; and his devotion to his profession was practically evidenced by the fact that recently, when at home on a holiday, he took out the course at the London School of Tropical Medicine.

Dr. Nightingale's retirement from Bangkok is much regretted by the whole of the foreign native community in the capital of Siam. He had thrown himself so heartily into the public work of the city, and had accomplished so much in the direction of public health improvements, that his retirement is looked upon as a public loss.

It was Dr. Nightingale who founded the Sanitary Department of Bangkok, which, during the past five years, has done so much for the sanitation of the city. He organised the office of the Port Medical Officer, established a system of quarantine and medical inspection on the most modern, *i.e.*, British, lines, with the result that plague has been completely warded off. When rinderpest raged throughout Siam, and the exportation of cattle to Singapore and elsewhere was threatened to be stopped, Dr. Nightingale took the matter in hand, and by a judicious and careful system of inspection was able to satisfy the requirements of the government of the Straits Settlements and other governments, and the trade in cattle—one of the most important—was allowed to continue. The gain to the community by his action proved of great commercial importance.

Dr. Nightingale was consulting physician at the palace, and the members of the royal family frequently availed themselves of his services. Of the many other public health and professional services

rendered by Dr. Nightingale, we may mention that he was medical officer in charge of the Bangkok police, the public abattoirs were erected under his supervision, slaughter-houses were controlled, &c., &c.

This long list of good work testifies to Dr. Nightingale's organising ability, his knowledge of public health, his tact in dealing with foreigners and natives, and we can well understand that the residents in Bangkok regret his retirement and look upon it as a public loss. The cause for his retirement was unfortunately due to his wife's state of health; but we are sure that wherever Dr. Nightingale settles, whether at home or abroad, he will make his mark and be welcome.

Obituary.

THE LATE PATRICK THURBURN MANSON, M.B.LONDON.

IN our last issue it was our painful duty to record the death of this young and promising physician, and with the present number of the Journal we issue his photograph taken shortly before leaving England for Christmas Island.

Patrick Thurburn Manson, son of Dr. Patrick Manson, C.M.G., F.R.S., LL.D., was born on August 20th, 1877, at Amoy, China. He received his early education at Dollar Academy, Perthshire, and Harrow. Amongst his schoolfellows he was held in high estimation for his athletic capabilities, and as a football player he was especially distinguished. He entered as a student at Guy's Hospital, London, and after holding several of the important clinical posts open to students at the Hospital he graduated M.B.London in 1900. During the summer session of 1901 he went to Aberdeen to study pathology under Professor Hamilton of the University of Aberdeen, and returned to London to work at the London School of Tropical Medicine. Whilst attending the course of Tropical Diseases he was attached to the Seamen's Hospital at the Albert Dock as one of the Resident Medical Officers, and did a great deal of medical, surgical and clinical work in the wards, and in the large out-patient department of the Hospital.

It will be remembered that in the autumn of 1900 Dr. Patrick Thurburn Manson was one of the two volunteers who submitted to be bitten by malaria-infected mosquitoes brought from the Roman Campagna. This experiment, initiated and carried out by his father, Dr. Manson, was undertaken for the purpose of conclusively proving the possibility of malaria-infected mosquitoes carrying infection to places far removed from the spot where they imbibed their dose of parasites. Young Manson readily came forward to be experimented upon and to test the truth of the doctrine of malaria-mosquito inoculation. All the world knows the result of that experiment: how that Dr. Manson and Mr. Warren were bitten by these mosquitoes from the Campagna, and how both after a short period of

incubation developed benign tertian fever. This was not the only point proved by this experiment, for some nine months afterwards Dr. Thurburn Manson had a recurrence of fever, an account of which was communicated by him to the *British Medical Journal*. Several such recurrences took place, some of which were of marked severity. The peculiar latency of malarial infection in a non-malarial country was thus proved as a scientific fact. These recurrences of fever, although very severe at the time of the attack, had no effect upon young Manson's fine physique, and he threw off the effects of the paroxysms of fever quite readily.

His father, Dr. Manson, had long contemplated a thorough investigation of one of the most deadly scourges of certain tropical countries, namely, Beri-beri. It is only those familiar with the Far East who can grasp what Beri-beri means. To the medical man its presence has long been a puzzle and its cure by drugs an impossibility. To the merchant and employer of labour the ravages of Beri-beri have proved ruinous to many an enterprise; and British traders in the Malay Peninsula, in the Malay Archipelago, as well as the Dutch in Java and Sumatra, know but too well what terrible havoc it plays in plantations, on board ship, and wherever it attains a hold.

Dr. Manson has long urged the necessity for a scientific investigation of this disease, and when Beri-beri broke out in Christmas Island, off the coast of Java, he turned his attention to the possibility of sending out a commission to deal with the matter. Dr. Durham, of the Liverpool School of Tropical Medicine, was selected as one of the commission. Than Dr. Durham no more suitable investigator could have been found. Although just returned from the West Indies, where he nearly lost his life from yellow fever which he had gone to investigate, he nerved himself for yet another and more distant journey, and proceeded to Christmas Island in November, 1901. His *confrère* was P. Thurburn Manson, who by his education, his training, his keen scientific spirit, and enthusiasm for investigation, was eminently qualified to undertake the laborious and self-sacrificing task. We know the rest: how shortly after his arrival at Christmas Island he met with the accident which terminated his life, and how the sad news was received in this country. These we referred to in our last issue, but it will be a long time before the shock of the news of his untimely end subsides, and as long as science and her findings are revered so will the name of Dr. Patrick Thurburn Manson live, and his memory will ever continue green amongst us as one of those who laid down his life for others.

TREATMENT OF BILIOUS HÆMOGLOBINURIC FEVER.—Dr. Paucot recommends subcutaneous injections of 200 to 300 cc. of chloride of sodium, 10 per 1,000, in the treatment of bilious hæmoglobinuric fever. Of seven cases thus treated by Dr. Paucot six recovered. —*Arch. de Méd. Naval.*, Sept. and Oct., Paris, 1901.

THE MALARIA EXPEDITION TO NIGERIA.

THE published reports of the Expedition sent by the Liverpool School of Tropical Medicine to study Malaria and other Tropical Diseases in Nigeria have been for some time before us. A careful study of these reports bear testimony to the care, labour, and scientific acumen and enthusiasm of the several members of the Commission, and their work abounds with a number of real additions to our knowledge.

The objects of the present Expedition were as follows:—

(1) To further explore West Africa to ascertain under what varied conditions mosquitoes of the genus *Anopheles* lived and propagated, with a view of ascertaining the most feasible and practical methods of preventing malarial fever.

(2) To investigate the conditions under which malarial fever is conveyed to Europeans.

(3) To corroborate and extend recent discoveries and researches on the subject.

It was not intended to limit observations to malarial fever alone, but to study also other tropical diseases as opportunity arose, and to note in addition the general sanitary condition of the places visited.

History of the Expedition.—Nigeria, Northern and Southern, were chosen for the field of operations.

The expedition consisted of:—

H. E. Annett, M.D., D.P.H.(Vict.), Demonstrator in Tropical Pathology, Liverpool School of Tropical Medicine; J. Everett Dutton, M.B., B.Ch.(Vict.); J. H. Elliott, M.D.(Toronto).

One of the most interesting investigations undertaken by the Commission, and which we here reprint, was in connection with

THE RELATION BETWEEN *FILARIA NOCTURNA* AND *FILARIA DIURNA*.

The many points of resemblance between the embryos of these two worms suggest the question of their identity, and in favour of the view of their identity many facts can be brought forward. In consequence of the importance of the subject, and the many points of interest involved therein, we propose to treat of the arguments for and against in some detail; and to arrange them under some chief headings.

Geographical distribution.—As has been already pointed out, the distribution of elephantiasis (caused by the presence of the adult form of *F. nocturna* in the lymphatic vessels and other sites) is extremely wide; but limiting ourselves to the distribution of *F. nocturna*, as determined by the presence of embryos in the blood, it corresponds in certain regions with that of *F. diurna*—the two occurring side by side throughout large tracts of country. On the other hand, however, there appear to be many lands where *F. nocturna* alone is found; but as far as is at present known, in no district has it been shown that *F. diurna* prevails alone. Reference must again be made in this connection with the conditions occurring in some of the Islands of the Pacific, already mentioned, where elephantiasis is very prevalent, and an embryo occurs in the blood of many natives, which resembles very closely *F. nocturna*, yet shows none of its characteristic periodicity.

The microscopical appearances of the embryos.—It has already been stated that in West Africa we were unable to distinguish the embryos in the blood of natives infected with *F. nocturna* and *F. diurna* respectively, by any means whatever. They appeared identical in their appearance, characters, measurements and movements in fresh preparations, and correspond in length, breadth, staining reactions, and in the possession of the same number of "spots," situated at similar points along the length of the worm and of the same shape and size. The sheath, a common feature of each, appeared identical. Moreover, the West African *F. nocturna* resembles very closely that of China and India as described by Manson.

The numbers in peripheral blood.—Here, again, there is a close similarity between the two worms. An ordinary case of either infection presents from twenty to sixty embryos in a drop of blood from the finger, at the time when the maximum number is present in peripheral blood—although in each case so many as four to five hundred may be present in exceptional infections.

Periodicity.—It was this phenomenon, and this alone, which led Manson to regard *F. nocturna* and *F. diurna* as distinct species. And certainly, in the limited condition of the knowledge of the subject, it was a very natural conclusion, one large set of cases which had been examined showing a characteristic periodicity with a maximum number of embryos present in peripheral blood at midnight, and a smaller set presenting the reverse conditions, a maximum number at midday. The departure from this interesting regularity to be first noted was recorded by Thorpe in the Tonga Islands, where a large percentage of the adults showed symptoms of elephantiasis, and where an examination of a large number of natives proved the presence of embryos in their peripheral blood both during the day and during the night in approximately equal numbers, and moreover showed that the embryos were present through the whole of the day.

We have already given details of several cases illustrative of the same conditions, and furthermore we have shown that cases of filarial infection occur in whom the hour at which the maximum number of embryos is present in peripheral blood is not midday and midnight, but may be any other hour—3, 6, or 9 a.m. or p.m. And besides we have shown that "pure" cases of *F. diurna* and *F. nocturna* are considerably less frequent in West Africa than these irregular cases.

The definitive hosts.—Thorpe, probably bearing in mind the classical experiment of Mackenzie, and the repetition of that experiment in another case by Manson, by which it was proved that by a change in the habits of a case of *F. nocturna*, the periodicity of the embryos could be completely inverted, becoming thus similar to that of *F. diurna*, explained the peculiar phenomenon of the occurrence of the embryos in the blood of the natives of the Friendly Islands by the habits of the natives, which he thus describes from Mariner's classical account of the Tonga Islands.

"The natives employ themselves in conversation not only at any time during the day but also at night. If one wakens, and is not disposed to sleep again, he wakens his neighbour to have some talk. By and by, perhaps they are all roused, and join in the conversation. It sometimes happens that the chief has ordered his cooks in the evening to bake a pig or some fish and bring it hot in the middle of the night with some yams. In this case the torches are lighted, and they all get up to eat their share, after which they retire to their mats; the torches are put out, some go to sleep, and others talk perhaps till daylight."

Similar habits are in practice among the natives of the whole of West Africa, but to a larger extent and on a larger scale. We were often told by natives from different parts of the Coast that it is a common practice in the respective countries to which they belong to sing and dance the whole night through, especially on moonlight nights. In fact we have ourselves heard the midnight orgies in the native towns which we visited, and especially of the Kroo boy gangs in the towns of Southern Nigeria. Moreover, we often observed, especially in those towns where civilisation was very backward, the natives asleep during the middle hot part of the day; indeed the Kroo boy in English Government employ steals a midday nap whenever he can. These habits have been practised, no doubt, for generations, and probably were prevalent to a much greater extent for years before the influence of Europeans was felt. Such conditions would, in a great measure, account for the variety in the cases of filarial infection we met with in West Africa, and which Thorpe observed in the Friendly Islands, and point strongly to the identity of the two embryos, or rather to the phenomenon of the accommodation of the one or the other or of an original embryo perhaps exhibiting no periodicity whatever, to the varying habits of the natives who formed their habitat.

The intermediary host.—*F. nocturna* has been successfully cultivated in several species of mosquitoes of both genera. In West Africa, after several attempts, we were able to cultivate this embryo in *Anopheles costalis*; but all our efforts to cultivate *F. diurna* failed. But this is not remarkable, for, if *F. diurna* had been evolved in consequence of the habits of the natives, it is not unnatural to expect that its intermediary host is an insect, probably a mosquito, not essentially nocturnal in its habits such as *A. costalis*, but one whose habits are diurnal.

Analogy with avian filariasis.—In the chapter on avian filariasis we describe eleven new species of filariæ, each having a different embryo; in fact, we were soon able after a little practice to decide the species of the worm even by a study of the stained specimen of the embryo. Each species then possesses distinct adults, which give rise to a characteristic embryo. This would suggest a similar condition among human filariæ, and thus that *F. diurna* and *F. nocturna*, being indistinguishable in fresh and stained specimens, have a common adult form.

The adult form.—The adult of *F. nocturna* is well known—*F. bancrofti*. The adult of *F. diurna* has

not yet been described, unless *F. loa* be that form. Now, the distribution of *F. loa* is, as far as we can ascertain, limited to the West Coast of Africa, and Manson makes the same statement. It has not been met with in any other part of the world,* and the occurrence of a worm of the length of *F. loa* occurring under the conjunctiva of the eye, cannot possibly have been overlooked anywhere.

F. diurna, as far as we at present know, is also apparently limited to the West Coast of Africa, and has been found in some cases of natives in which *F. loa* has been removed from the eye—although this is not remarkable as anything more than an ordinary coincidence, considering the prevalence of *F. diurna* cases on the coast. Moreover, cases of *F. loa* have occurred in which no embryos could be demonstrated in the blood.

The conditions in the Friendly Islands, previously often referred to, may perhaps be quoted as an exception to the statement above—that *F. diurna* is limited in its distribution to West Africa—since the embryos cannot be regarded as nocturnal. Probably this condition will be found to be much more extensively distributed. On the other hand we have described the embryos of *F. loa* as very similar to those of *F. nocturna*: but on closer study some points of difference may be noted in the disposition and number of the spots. Such a close resemblance indicates either that they are identical with *F. diurna*, and that, therefore, *F. loa* is the parent form of *F. diurna*, or that, being very much alike in all other respects in the matter of the spots as just mentioned, they are intended for a more or less similar life history in their intermediary hosts.

To sum up, although the weight of evidence is on the side of the identity of *F. nocturna* and *F. diurna*, there are many points which remain to be cleared up before the question can be settled. The *F. loa* has introduced a serious difficulty into the subject, and it appears to us that a solution of the mystery can only be obtained when the embryos in a pure case of *F. diurna* have been successfully and completely cultivated in their intermediary host—which is still to be discovered—to the final larval stage, and perhaps it may become necessary to perform experiments of infection of man by the use of infected intermediary hosts before a complete solution is procured.

DR. W. E. LLOYD ELLIOTT'S ARTICLE ON PANI-GHAO.

THE Editors wish to notify that the excellent article on "Pani-ghao, the Water Itch or Sore Feet of Assam Coolies," which was published anonymously in the JOURNAL OF TROPICAL MEDICINE, December, 1900, was communicated by Dr. William E. Lloyd Elliott, M.D., Medical Officer of the Assam Frontier Tea Co., Dibrugarh, Assam. This article is referred to by Dr. A. B. Dalgetty in the JOURNAL OF TROPICAL MEDICINE for March 1st, 1901, and in a foot-note the Editors

* Stossich states that it occurs in the Antilles and Guiana, but Manson says, in the latest edition of "Tropical Diseases," 1900, "It is peculiar to the West Coast of Africa."

explain how the paper came to be published "anonymously."

The Editors wish to express their regret that an article of such high merit and indicative of such close scientific study should have been published without Dr. Elliott's name being appended to the manuscript.

Article for Discussion.

THE DANGERS OF SUBCUTANEOUS INJECTIONS OF QUININE.

By AUBREY HODGES, M.D.Lond.
Medical Officer, Uganda Protectorate.

THE very great therapeutic value of this form of administration of quinine is, I suppose, generally acknowledged, and in offering the question of its dangers for discussion, the JOURNAL OF TROPICAL MEDICINE implies the importance of collecting the varied experiences of medical men in its use, and also the advantage likely to be gained by careful study of such untoward accidents as, from time to time occurring, may act as an unfortunate deterrent from its confident and prompt employment.

If statistics could be collected of these accidents (abscess, slough, tetanus) which have followed subcutaneous injection of quinine, with details as to the precautions taken, the salt used, the site chosen, and the condition of the patients injected, they would probably form a step towards the explanation of their occurrence, and possibly a guide for their avoidance in future.

I have been in the habit of employing hypodermic injection as a routine treatment in all bad cases of malaria, in blackwater fever, and in such milder cases as did not yield readily to the administration of quinine by mouth, and hitherto I have derived nothing but encouragement from the practice. I do not know, of course, how soon it may be my misfortune to be confronted with an abscess or a slough, but up to the present this has been spared me, and I have seen no worse mishap than a little inflammatory swelling which has disappeared after forty-eight hours at the farthest.

It having been formerly my lot to inject large quantities of diphtheria antitoxin, I have followed with quinine as nearly the same method as circumstances would allow. Whether my freedom from accidents has been due rather to the constitutions of my patients or other causes than to the manner of my procedure I cannot say. My method is very simple, for one works (out here) under somewhat primitive conditions. But probably it does not differ essentially from others commonly in use. It is as follows:—

First, I have a soup-plate scalded and filled with boiling water. In this I place a tablespoon and a teaspoon which have previously been cleaned and boiled. Then, having boiled in a test-tube the water necessary for making the solution, I turn it out into the empty tablespoon, in which I triturate the salt or tabloids to be dissolved with the convex of the tea-

spoon, so as to ensure perfect solution and freedom from grit. I thus have the liquid in a receptacle convenient for taking it up quickly with the syringe, while the hot water in the plate prevents cooling and precipitation of the drug during the next step, which is to boil the needle, with the wire in it, in the test-tube. This done, and the wire removed, the needle is immediately plunged into the site chosen for injection (previously prepared) and remains there until the operation is finished, no matter how many syringefuls are required to complete it. Other soluble drugs, such as morphia, strychnia, or pilocarpin, may be added to the injection if needed at the time. If no test-tube is at hand the tablespoon may be used for boiling the needle, which is then straightway inserted, and the water for solution afterwards also boiled in the spoon. Both syringe and needle are carefully cleaned after operation as well as before, and I need scarcely refer to the care of one's own hands. I use, when possible, distilled water both for making the injection and for boiling the needle, but more often I have had to use ordinary filtered water, and sometimes I have been obliged to dispense even with filtration.

In my opinion the best needle for the purpose is a steel one with not too fine a bore, and probably a syringe to hold from 2 to 4 drachms would be more convenient than the ordinary hypodermic which I have hitherto used. I have employed either the hydrochloride or the bi-hydrochloride salt, of which I prefer the former as being less irritant; and I have also used the hydrobromide, but found it liable to block the needle and less easy to give in large doses. My maximum dose for one injection has been 30 grs. I have frequently given 15 grs., and more often 10 grs. or less.

There is no doubt in my mind that by far the best site for injection is the buttock, into the muscles of which the needle should be deeply plunged. I think, however, that this site is in a measure contraindicated, as also the scapular region, by emaciation of the patient, or previous prolonged confinement to bed. Next in order I prefer the abdominal connective tissue, where I inject in the same manner as diphtheria antitoxin is injected. Failing these, I use the scapular region, in which, however, I have found that there is more after-discomfort to the patient. I do not think that such parts as the arm or front of the thigh are suitable for quinine injection, and some of the abscesses of which I have known have occurred in such situations.

Apart from sepsis, the most obvious precautions seem to me to be able to avoid undue irritation and to minimise tension in tissues in which the circulating blood is itself the seat of wasting disease, in which adjustment and repair are for the time being at a low ebb, and the process of absorption is probably not at its best. Injections should not, I feel certain, be repeated in or about the same spot until all signs of local reaction from previous injection have disappeared. The three regions favourably noted above offer six different sites, which may be used in turn. Quinine solution is undoubtedly irritant, and becomes more so if acid has to be added, as in dissolving the sulphate, and the tension from the volume of liquid employed

is considerable if it is too rapidly injected, or if an unsuitable part is chosen. The fluid should be injected slowly, to allow of gradual distension of the tissues, and into a part which is capable of such distension without undue injury. Also I think the solution should not be too concentrated, and I therefore always add more water than is required to dissolve the drug.

It has seemed to me that the larger the dose the greater has been the reaction or irritation, but I have not found that an injection repeated at a spot where all swelling and tenderness had subsided gave rise to any more trouble than the original one.

In order to avoid too frequent subcutaneous injections of quinine, I have in many cases exhibited it in enema, and, in fact, have found it a very sound plan to begin by hypodermic injection, and afterwards, as far as possible, to keep up the effect by enema. I have been struck by the usefulness of the hydrochloride for this purpose. I have found quininism produced almost if not quite as quickly as if the drug had been taken into a healthy stomach. I give it along with the nutrient enemata (peptonised milk and meat essence) which are usually needed at the same time, and with it any other soluble drugs which may be necessary. The addition of a little brandy, which is often itself required as a stimulant, makes the quinine readily soluble at the proper temperature, and I have found little difficulty as to retention by the bowel, with proper care, even in the worst cases. I may mention that, in a case which I hope soon to report, a single $\frac{1}{2}$ -gramme dose in enema appeared to produce immediate hæmoglobinuria in a patient with a history of quinine idiosyncrasy.

Very likely irritation, tension and septic inoculation do not cover the whole ground of the causation of quinine abscess. I have heard anæsthesia at the site of injection mentioned as having occurred, apart from tissues about to slough. Is it possible that these abscesses may sometimes be in some way analogous to bedsores? Are they ever due to impaired nerve-influence caused by the action of the drug itself?

The whole work is evidently the production of a teacher, who not only knows his work, but knows how to teach. There is no redundancy in the text, yet no detail is left in confusion or to the imagination. To the physician in the tropics who has to take up investigation on his own behalf and with no one to advise him as to methods, this book must prove indispensable. All details necessary for preparing apparatus, for bacteriological methods, for recognition of bacteria, diseased cells and tissues by the microscope, &c., are clearly and intelligibly given.

Mr. Strangeways Pigg's book affords a complete and infallible guide, whereby the student and practitioner working alone can acquaint himself with the practical methods necessary for research in pathological investigation, or in any clinical work which he may have to undertake.

News and Notes.

DR. NEILD COOKE, Health Officer of Calcutta, has been re-appointed for a further period of three years. Dr. Cooke is coming home on leave this summer.

A PUBLIC memorial is to be raised to the late Surgeon-General R. Harvey, C.B., Director-General of the Indian Medical Service.

TURKISH officials require that all medicines imported into Turkey shall meet the requirements of the French pharmacopœia, and that all formulas should be written either in French or in Turkish. Quantities and proportions must be clearly stated, and the names of drugs must be given in purely scientific, as opposed to popular, nomenclature.

A PUBLIC health laboratory has been opened and fitted up in Bangkok under the direction of Dr. H. Campbell Highet.

Reviews.

CLINICAL PATHOLOGY AND PRACTICAL MORBID HISTOLOGY. By T. Strangeways Pigg, M.A., Demonstrator of Pathology in the University of Cambridge. Messrs. Strangeways and Sons, London. 1901. Pp. 167. Illustrated.

Mr. Strangeways Pigg has given us a most useful book. Compact, clearly printed, interleaved for notes, and illustrated, the book is eminently suited for practical work.

The Clinical Pathology of Blood is dealt with in thirty-one pages; four pages are devoted to the sputum; and the subject of urine extends over sixteen pages. Diphtheria, ringworm, gonorrhœa, and pus receive special attention; the clinical diagnosis of each by microscopic methods being carefully gone into. Practical Morbid Histology occupies the last twenty-two pages of the book.

Current Literature.

BEEF-WORM IN THE ORBITAL CAVITY.

By THOMAS W. F. GANN, M.R.C.S.Eng., L.R.C.P.Lond., L.S.A.

District Surgeon, Corozal, British Honduras.

To anyone who has lived for any length of time in either Central America or South America the parasite known as the "beef-worm" must be familiar either from hearsay or possibly from painful personal experience, as, though fortunately not of common occurrence, it is one of the most distressing and painful of the innumerable insect pests of these regions.

The creature starts in life as an egg, which being laid by the parent fly beneath the human skin or mucous membrane soon hatches out into a small worm, which rapidly increases in size and reaches in a few months a length of from one to two inches and a diameter of from three-eighths to three-quarters of an

inch. The worm is of a dull greyish-white colour, divided into from twelve to twenty segments and covered by stiff black hairs or bristles, according to its age, along the middle two-thirds of its body, each segment possessing a separate row of hairs completely surrounding its anterior and posterior margins. The tail end communicates with the exterior by a circular orifice surrounded by a prominent raised margin, the head end being deeply embedded in the tissues and supplied with two strong, semi-circular, black hooks, by means of which it attaches firmly, rendering its expulsion extremely difficult. It advances and withdraws its tail with a sort of rhythmic motion which gives rise to the characteristic sensation of "something moving and alive" within the swelling caused by it upon the skin or mucous membrane. The parts most affected by this parasite are the extensor surfaces of the arms and legs, the back, and shoulders and the chest; but exceptional cases have been recorded of its occurrence in the face and even upon the penis.

Certain individuals appear to be particularly susceptible to the worm, and if one of these should go into a district infested by it he is practically certain to become the host of one or more. Negroes are far more susceptible than are whites. Other persons, again, appear to enjoy complete immunity from the pests; they may go into a district swarming with the worms, sleep on the ground out of doors without a net for months at a time and never get a single one, though other persons of the same colour and nationality would under similar circumstances be literally eaten alive. The worm appears to be limited to small and clearly-defined districts within which a man may, if he sleeps uncovered for a single night, be almost certain of contracting it, though less than a mile distant perfect immunity is enjoyed.

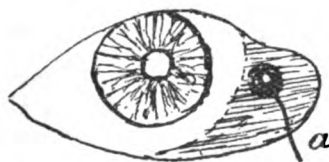


FIG. 1.—Rough diagram of the eye of the patient, the shaded portion indicating the inflammation and protrusion of the inner canthus. *a*, Circular aperture with prominent raised margin through which the tail end of the worm protruded from time to time.

The present case of a beef-worm in the orbit is, so far as I am aware, unique. The patient, a Spanish lad, about 18 years of age, came to me on September 16th, 1901, stating that he had been living for some considerable time in the bush and that during the last three months he had been suffering from pain and inconvenience in the left eye, together with a sensation as if something were moving about within the socket. On examination the eye was seen to be a good deal more prominent than its fellow; the conjunctiva was more or less congested throughout its whole extent, and the entire inner canthus of the eye was enormously bulged forward. Immediately to the inner side of the caruncula lacrymalis was a circular opening about one-twelfth of an inch in diameter, surrounded by a prominent rim, and at the bottom of this opening

appeared a small, whitish knob which from time to time was slightly protruded beyond the opening, but if touched it was immediately withdrawn so far back as to become almost invisible (fig. 1). The negro method of treating the worm is first to cover the hole over with a plug of wet tobacco for a day or two, and then with great force to squeeze it out, the tobacco appearing to make the creature lose its hold with the hooks attached to its head. This method was obviously impossible in the eye, so I injected a few minims of a

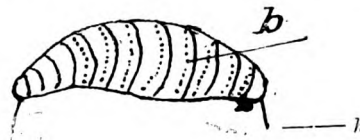


FIG. 2.—Rough diagram of the beef-worm. *h*, Head, showing two short black hooks with which the worm affixes itself to deep tissues; *t*, tail; and *b*, bristles. These bristles are set in rows around each segment of the middle two-thirds of the worm.

strong solution of tobacco into the aperture with a hypodermic syringe and then covered it over with a plug of moist tobacco-leaf and left it for a couple of hours. On removing the tobacco-plug I found the creature much less active, and after some little trouble I managed to seize the tail-end with a fine forceps and little by little to withdraw the whole worm. The patient made a good recovery and did not lose the sight of his eye.

I enclose a rough sketch and two photographs of the worm (figs. 2 and 3), as well as the creature itself



FIG. 3.—(a) The ventral aspect, and (b) the left dorsal aspect of the actual worm; life-size. From photographs.

preserved in alcohol; it measured when first extracted one and three-eighths inches in length by three-eighths of an inch in diameter and was covered over the middle two-thirds of its body by short dark bristles. It is only about half-grown, as in the full-grown worm these bristles attain a length of half an inch or more.

The curious point about this case is how the fly managed to lay its egg in such an exceedingly sensitive spot as the inner canthus of the eye without the sufferer becoming aware of the fact.—*Reprinted from THE LANCET, January 4th, 1902, by permission of the author.*

PLAGUE.

PREVALENCE OF THE DISEASE.

INDIA.—During the two weeks ending February 22nd and March 1st, there were 12,675 and 21,789 deaths from plague in India. The enormous increase during the latter week is accounted for by a general rise in the death-rate from plague in Northern India. In the Punjab alone 10,525 persons died of plague

during the week ending March 1st. Plague has broken out in Hyderabad (Scinde); between 40 and 50 deaths daily were reported in this district during the first days of March. Between February 19th and March 19th, 50,000 persons died of plague in the Punjab. In Calcutta, during the four weeks ending March 15th, 1,488 persons died of plague.

MAURITIUS.—During the weeks ending March 13th and 20th, the number of deaths from plague in Mauritius numbered 7 and 3 respectively.

CAPE OF GOOD HOPE.—On the 1st March but one case of plague remained under treatment in any of the plague hospitals in Cape Colony. No fresh cases were reported during the month of February.

LEPROSY.

THE PRIMARY LESION OF LEPROSY.—From time to time the question of a primary lesion in leprosy is brought up for discussion and remark. L. Glück, of Bosnia, in the *Wien. Med. Woch.*, of July 27th and August 3rd, 1901, returns to the subject and describes the initial leprosy lesion. Glück, however, states that this lesion is but part of the initial eruption, and therefore can in no sense be termed a primary local lesion such as the primary sore in syphilis. We find this confusion in statement repeated by several writers on leprosy, and we would especially draw attention to the difference between a primary lesion which is of a general eruptive character, and therefore characteristic of general infection, and a primary lesion in the sense of a local sore. That some writers believe they have discovered a local primary infective sore is well known, but confirmation of the matter, even although supported by fairly positive evidence by Sticker, Arning, Daubler, Lutz, and Geill, is by no means conclusive. Lutz and Geill, from observations made in the Dutch East Indies, state that 50 per cent. of the cases of leprosy seen by them appeared in the feet. Sticker, on the other hand, holds that the nose is more often the site of the primary lesion of leprosy. Given a local primary lesion, the question of its removal is one which becomes of surgical importance. So far as excision of the so-called primary lesion of leprosy goes, we have it on the authority of von Bergmann that the excision does not prevent the subsequent appearance of general leprosy. Whether von Bergmann was dealing with a real primary local sore or only with the earliest lesion of the initial eruption is not known. Further exact information on this point is wanted, and although it is difficult to obtain it is not impossible. We are apt to neglect and set aside statements by natives and others of leprosy contracted by a single coitus with a leprous woman as being merely popular ignorance, without a single scientific argument in support of the rejection of the statement. There is much really to be said for the popular idea and it may be correct.

GANGRENE IN ELEPHANTIASIS.—A case of gangrene of the foot supervening upon elephantiasis of the right lower extremity was reported before the Brooklyn Surgical Society by Dr. J. Richard Kevin, in February, 1902. The patient, a woman, aged 22, had had the elephantoid condition since her tenth year, and since 1893, when the limb was amputated, the

patient has been married and had two healthy children. It is to be presumed that the elephantiasis is due to filarial infection, and it is interesting to note that the woman lived in the State of Ohio.

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Archives Russes de Pathologie, de Médec. Clinique et de Bacteriologie.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Brooklyn Medical Journal.
Caducée.
Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Esercito
Hong Kong Telegraph.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
Journal of the American Medical Association.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Medical Record.
Medical Review.
Merck's Archives.
New York Medical Journal.
New York Post-Graduate.
Pacific Medical Journal.
Polyclinic.
Public Health.
Revista de Medicina Tropical.
Revista Medica de S. Paulo.
Sei-i-Kwai Medical Journal.
The Hospital.
The Northumberland and Durham Medical Journal.
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- 1.—Manuscripts sent in cannot be returned.
- 2.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.
- 3.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.
- 4.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.
- 5.—Correspondents should look for replies under the heading "Answers to Correspondents."

The Journal of Tropical Medicine.

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In order to meet the constant enquiry for addresses of respectable firms catering for the various require-
ments so difficult to obtain abroad, we give a list of names and addresses which we trust will be found
useful to our numerous correspondents and subscribers.

Original Communications.

FILARIASIS IN ST. KITTS, W.I.

By GEORGE C. LOW, M.A., M.B., C.M.(Edin.).

Crags' Scholar, London School of Tropical Medicine.

THE island of St. Christopher, more popularly known as St. Kitts, one of the West Indian Colonies classi-
fied under the group of the Leeward Islands, lies in
lat. 17° 18' N. and long. 62° 48' W., in the vicinity of
Antigua and Montserrat.

It measures about 28 miles in length by about 5
miles at its greatest breadth and consists of a central
core or ridge of volcanic mountains, the land sloping
more or less abruptly from this to the sea. The soil,
which is chiefly covered with sugar cane cultivation,
is sandy and porous, and quickly absorbs large quan-
tities of water, any excess being carried off by large
watercourses to the sea; these in the winter months
are dry, and even after the heavy rains of summer
they only flow for three or four days.

Basse Terre, the chief town of the island, lies on the
leeward coast, on flat, sandy ground, near the base of
the end of the ridge of hills, and is typically West
Indian, native huts and dwellings being mixed up in
close contiguity with the houses of the better-class
white inhabitants. Malarial fevers used to be com-
mon in the town, but since the "pond pasture," a
piece of swampy ground lying immediately to the
south, was filled up, they have almost entirely disap-
peared.

PREVALENCE OF FILARIAL DISEASE.

Filarial diseases are very prevalent in the island,
the percentage of infections being higher than that in
any of the other islands I have examined.

One is at once struck, even without special exami-
nations being made, by the fact that large numbers
of the inhabitants are suffering from elephantiasis, or
what is known locally as "Barbados legs," and the

medical men practising there also state that the dis-
ease is very common, many cases being annually
treated in the general hospital and in private.

On the occasion of my visit to the island, the usual
procedure for accurately determining the amount of
filarial disease was adopted, namely, nightly blood
examination of as many people as possible, selection
being avoided by taking indiscriminately white people
of the better classes, healthy individuals from the
streets, and cases in the Colonial hospital suffering
from ordinary complaints. Any cases suffering from,
or being treated for, filariasis *per se*, were considered
separately for the study of the various features of the
disease.

In this way the blood of 143 individuals was
examined at night, and no fewer than 47, or 32.8 per
cent., were found to be suffering from filariasis. Nine
of these, though being treated for various complaints,
showed pathological signs of filariasis as well; the
other 38 showed no appreciable symptom of that con-
dition, the diagnosis being arrived at solely by finding
the embryo of *F. nocturna* in their blood.

This percentage, as I have already stated, is very
high for the West Indies, for Barbados, where the
disease is supposed to be very common, has only
12.66 per cent. of the people infected, and the hilly
islands of St. Lucia and Dominica, 7.58 per cent.
and 7.63 per cent., respectively.

TYPES OF THE DISEASE.

Filarial lymphangitis, locally known as "fever and
ague" and "rose," is very common, and very few
of the white inhabitants have escaped attacks at some
period or other of their lives. Chyluria, Dr. Christian
Branch informs me, is rare, but lymph scrotum,
filarial abscess, varicose groin glands, and elephan-
tiasis are all very common. Many types of the latter
disease are seen, affections of the lower limbs prepon-
derating, and some of those are of an enormous size.
In addition, cases of elephantiasis of the scrotum,
prepuce, penis and breast occur, and in one girl, the

pedunculated groin elephantiasis, described by Corney in Fiji, and Daniels in British Guiana and Fiji, was very marked, the condition being associated with a permanent thickening of the thigh as well.

Many cases with swarms of embryos in the peripheral blood at night showed no sign of any disease, and this class, as the statistics bring out, forms the large majority of those infected.

SOME POINTS ON THE PATHOLOGY OF THE DISEASE.

Embryos in Elephantiasis.—As many blood examinations of elephantiasis as possible were made, and out of a total of 14 typical cases, embryos were found in the peripheral blood of three, a most unusual thing, as may be seen from the fact that up to this time, out of 56 cases (Barbados 18, Trinidad 23, Demerara 15), embryos had only been found once, and this was in the case of a woman from the latter colony, Demerara, where filariasis is very common.

This bears out very completely Dr. Manson's statement "that it is not usual to find filaria in elephantiasis cases in the blood or elsewhere, *unless* it be in countries in which filariasis is very common and re-infections, or extensive infection highly probable" ("Tropical Diseases," Manson, Second Edition, p. 505).

This fact might be made use of in rapidly determining the presence of infection in any given place, *i.e.*, if one examine a series of elephantiasis bloods and find embryos present in several, it is a certainty that there will be a large amount of the disease (filariasis) in the place from which they come.

Absence of Embryos after Filarial Lymphangitis.—It is an interesting fact, and one that may help to elucidate some of the obscure pathological changes in this disease, that the night blood of a woman in the Colonial hospital at St. Kitts, who had just recovered from a first attack of filarial lymphangitis, contained no embryos, a similar condition having been noted four times previously in Barbados. The subsequent history of three of the latter was followed for some time very carefully, and blood examinations repeated many times always gave the same negative results. This must mean that in such cases the mature worms have either died, this probably causing the attack, or, owing to the blocking of the lymphatic in which the adult is lying, the embryos can no longer get into the blood stream. Whether or not fresh infection would be required for another attack is difficult to definitely determine, as old cases of filarial lymphangitis, that is, cases that have suffered before, go on having repeated attacks, two, three, or more in a month, even without any embryos being found in the peripheral blood. It is certainly not easy to imagine such individuals being so continually reinfected.

Post-mortem examinations might help to clear the matter up, but the chances of such must be very rare, as people do not die often just after an attack.

"*Post-mortem notes of a Filarial Case.*"—I was fortunate to obtain an autopsy on a man in Barbados, who during life had a few embryos in his peripheral blood at night, and as the notes are interesting, especially as they corroborate Dr. Manson's results obtained in London, they may be incorporated in this paper.

The man, a negro, had never suffered from any

filarial symptoms, but was the subject of a sarcoma which eventually caused his death, the latter event taking place one morning at 10 a.m.

No adult worms were found, though a prolonged and careful search was made; the thoracic duct was free, as were also the pelvic lymphatics, and there was no sign of any varicosity.

The fact of there being so few embryos in the peripheral blood pointed to only one or two adult worms and these of course may have been in any of the lymphatics.

Drops of blood were taken from the various organs in a similar manner to that adopted by Dr. Manson in his autopsy made in London and the results were as follows:—

| Organ | No. of Slides | Aggregate No. of Filariae | Average per Slide |
|-----------------------------|---------------|---------------------------|-------------------|
| Lung | 4 | 65 | 16.2 |
| Liver | 4 | 0 | 0 |
| Spleen | 4 | 0 | 0 |
| Kidney | 4 | 0 | 0 |
| Pancreas | 4 | 0 | 0 |
| Heart Muscle, right side .. | 4 | 0 | 0 |
| " " left side .. | 4 | 0 | 0 |
| Inferior Vena Cava | 4 | 0 | 0 |
| Aorta | 4 | 0 | 0 |

No embryos, it will be seen, were found, except in the blood-vessels of the lungs, and this is in all probability their special seat of selection by day, other neighbouring vessels and organs being inhabited as well when the infection is a large one and the embryos very numerous.

CAUSE OF THE DISEASE.

The cause of the disease and its spread by suitable species of mosquitoes has now been so completely worked out, that it is needless entering into its details again here.

It is sufficient to state that *Culex fatigans* abounds in St. Kitts, suitable places for its breeding existing in abundance. It is much more common than *Stegomyia fasciata*, agreeing in this respect with the island of Barbados, whereas in St. Lucia and Dominica the reverse is the case. The relative numbers of the two species of mosquito probably have to do with the degrees of prevalence of filariasis in these places.

PREVENTION OF THE DISEASE.

The prevention of filariasis, which really amounts to a careful personal prophylaxis by means of the mosquito net, and the destruction of the breeding grounds of *Culex fatigans*, the domestic mosquito, should be an exceedingly easy task for the island of St. Kitts, as the town of Basse Terre is small, and has now the advantage of having water laid on in pipes. The necessity of storing water in tanks and other receptacles, therefore, no longer exists, and such arrangements can easily be done away with.

Another main source of these pests is found, however, in the privy system, which is used throughout the

town. The only proper method of dealing with this is to abolish the system altogether for the dry earth one, a substitute I have strongly advocated to the Government.

Other collections of water, such as barrels, tubs and the like, can easily be dealt with by the Sanitary Inspectors when going their daily rounds. Fountains and ornamental tanks for growing water lilies in should have gold fish or minnows introduced, as these quickly eat up any larvæ present.

It is astonishing how few are the people in the West Indies who use mosquito nets; this carelessness undoubtedly greatly helps the spread of the disease. All the white people of St. Kitts in whose blood I found filarial embryos had never done so, whereas four individuals who had always been most careful in this respect had never shown any trace of filariasis and had no evidence of it in their blood. The figures I possess are perhaps too small to warrant any definite conclusion, but there is no doubt that a more careful personal prophylaxis would soon tend to diminish the disease.

St. Kitts has awakened to a sense of its danger and with praiseworthy energy has taken up the subject in earnest. It will be an interesting study in the course of the next generation to watch the gradual reduction and stamping out in the island of this loathsome complaint.

THE DURATION OF THE LATENCY OF MALARIA AFTER PRIMARY INFECTION, AS PROVED BY TERTIAN OR QUARTAN PERIODICITY, OR DEMONSTRATION OF THE PARASITE IN THE BLOOD.

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(Translated from the Italian by St. Clair Thomson, M.D.-London, F.R.C.S.)

PREFACE.

IN the years 1899, 1900, and 1901, Professor Ballori, the Director-General of the Hospitals in Rome, instituted in the several hospitals a special department for the patients suffering from malaria, in order that the very large amount of clinical material in malaria, which is met with every year in the Roman hospitals, might be studied with greater profit.

This special department was entrusted to my care, and I endeavoured to fulfil the task as completely and as rigorously as possible; I seized the favourable occasion for carrying out various studies which I had had some time in mind, on the subject of malaria.

Before entering on the present publication, I feel it a duty to offer my very warmest thanks to Professor Ballori both for the honour of entrusting this department to me, and also for having on every occasion furnished me with the necessary means in order to do the work thoroughly and bring it to a satisfactory conclusion. I hope that his confidence

has not been altogether misplaced, since I see that the results I have already arrived at have been thought worthy of appearing in the very important *Atti della Società per gli studi della malaria*, 1901, vol. ii., p. 109, &c.

Also I would express my thanks to Professor Giulio Bastianelli, Chief of the Section in which I particularly made my observations. I would also like to place on record the kindness and politeness of each and all of the physicians of the Hospital of S. Spirito, who always favoured me with notes on the patients I had under observation in their sections; and I would particularly mention Dr. Scipione de Rossi, who often came to my aid with wise counsel, and also Dr. Alphonso Cortini, who gave me his patient and cultured assistance. I would also thank for their assistance the physicians of the Ambulanza della Croce Rossa, to many of whom I am indebted for the following observations being complete and exact.

Chapter 1.—Introduction.

During the years 1899, 1900, and 1901, when studying the malarial patients in the Hospital of S. Spirito in Rome, I was able to collect numerous observations.

Of the results obtained from my observations of various points I have already published some of them, while others are in course of publication.*

In this memoir I intend to record the observations instituted on the duration of the latent period of malaria after the first infection, an investigation of much importance, but which up to the present has presented numerous uncertainties and contradictions between authors.

For this purpose it is necessary to refer to the standard which has guided me in differentiating cases of relapse from those of true primary infection. All my observations are based on considering as relapses "the successive reappearance of one or more infections which, without ever becoming cured, are reproduced at various intervals, the epidemic having ceased during all the healthy months, continuing until the new epidemic year or even longer, sometimes for several years" (Celli, *Atti della Società di studi sulla malaria*, 1901, vol. ii., p. 88).

This was the criterion that guided me in the present work. Now I would describe briefly the method on which I conducted my researches on the subject.

Chapter 2.—Material and Methods of Study.

From every patient with malaria who entered the hospital I carefully collected both the previous and recent history, trying to secure the most minute points upon previous illnesses, on the localities where the patient had contracted the infection, on its duration, and on the quality of the fever. To verify the dates of the progressive infections I found great

* *La Febre Quotidiana Estiva—Quotidiana Vera*—Pel Dottor Attilio Caccini, *Policlinico*, 1901.

Alcune osservazioni sull'epidemiologia della malaria, Dottor A. Caccini, *Policlinico*, 1901.

Sul comportamento dell'epidemie malariche, 1900-1901, studiate allo Ospedale di S. Spirito in Roma. Dottor Caccini, ecc., ecc.

assistance in the clinical records of the hospital, for a good number of the patients had been treated on previous occasions in the Hospital of S. Spirito. Often the records of other hospitals outside Rome were of great assistance to me, when the patient mentioned the city where he had been treated. In this way I was able to ascertain in every case the date and duration of the progressive infections, as well as their quality. Often I was also able to secure exact notes of the treatment they had had.

I found besides that the information furnished by the patients themselves was almost always exact, especially in regard to the quality of the infection, and to the periodical reappearance of the febrile attacks. In this way, by means of the history, I was able to follow the patient accurately, either during his infection, or during the relapses. I devoted special attention to the various treatments received, to the quality of the remedies, and to their method of administration—especially in regard to quinine—to methods of life, to conditions of habitation and locality, and to occupation or profession. In this way I was able to find out satisfactorily the part played by each in the renewal of new infections, and also particularly in the renewal of febrile attacks and relapses. I also endeavoured to ascertain what influence on the renewal of the fever was effected by the atmospheric variations, either by themselves or in relation to the occupation of the patient (cold, heat, moisture, dryness, rain, barometrical variations).

I took particular note of the type of fever in each infection and relapse, of the duration of each successive apyretic period, whether under the influence of quinine or on the spontaneous exhaustion of the fever.

I kept in mind that an infection or a relapse may pass unobserved for some time from the fact that it presents irregular symptoms (malaise, weakness, sweats, headaches, gastro-intestinal disturbances, hæmorrhage, &c.) without elevation of temperature.

To all these observations I added the systematic observation of the blood (which I repeated several times for every patient in both fresh and coloured preparations) and the study of the temperature curve and the clinical progress.

There were specially brought to my notice many individuals who were hospital visitors at S. Spirito every time that the fever seized them. I was able to follow certain of them for quite three consecutive malarial years. Contemporaneously with all these hospital researches I carried out other observations in certain localities in the Roman Campagna, which the facts I was gradually collecting indicated to me as places of particular interest for the study of the relapses. In this way I followed my patients from the hospital into the fields, and availing myself of the kind consent of the Director (Professor Ballori) I submitted these patients, where it seemed to me indicated, to treatment either by quinine, arsenic, or iron. Of these patients certain of the more intelligent voluntarily presented themselves for observation, and came at fixed periods to the hospital to be examined and to report to me on their companions during the intervening period. Most usefully help-

ful were the notes furnished to me by the doctors of the communes, and from the doctors of the Ambulances which the Croce Rossa had stationed in the vast Agro Romano. Almost all the cases were studied along with their temperature charts, and in association with the cycle of development of the parasite in the blood.

Chapter 3.—General Survey.

Of the patients studied in the three years, 1889 to 1901, I limit myself to reporting the facts collected between June 1st and December 31st for 1900, and from June 1st to November 15th of the year 1901. These will be found in the following table. Numerically the figures of these patients seem to me sufficiently convincing, hence I refrain from adding the cases collected in 1899, limiting myself to referring only to such of these as, from their special behaviour, presented anything particularly characteristic. In regard to 1901, the observations are still continuing, but in any case I limit myself to detailing, as I have said, those observed up to November 15th. I will report those observations since that date which appear to me to be worthy. It is well to note that I have absolutely excluded all the patients who gave negative results by the microscopical examination of the blood, and in whom the observations for some reason or other could not be completed, or who presented mixed infections. The following is the record of the cases of malaria considered by me in the period recorded.

Summarising it, we have:—

(a) Benign tertian—764 cases in 1900; 1,256 cases in 1901. Altogether 2,020 cases, of which 1,086 were regarded as primary (of these a good number came under observation during relapses), and 934 regarded as relapses.

(b) Malignant tertian—2,325 cases in 1900; 1,379 cases in 1901. In all, 3,704 cases, of which 2,275 were regarded as primary, and 1,429 as secondary.

(c) Quartan—415 cases in 1900; 440 cases in 1901. In all, 855 cases, of which 525 were primary, and 330 relapses.

Chapter 4.

In view of the enormous amount of material I have felt the necessity, in order to facilitate explanation and analysis, of making many classifications. I will therefore proceed to dilate upon certain constant data which impressed themselves on me from the first year of observation, and from which I have been able to draw up the following conclusions:—

(1) When an individual has been attacked by malarial fever, however it is treated it may happen that after a certain latent period the fever reappears.

(2) The relapse may take place (a) after a short interval, (b) after a long interval.

(3) In the relapses with short interval, the manner in which the clinical treatment of the primary infection has been carried out has a very great influence on the relapse itself, or at least on the number, gravity, and nature of the relapses.

(4) In the relapse, after a long interval, we constantly find, as determining factors of the relapse, the intervention of certain accidental factors which, on the other hand, in the relapses after a short interval,

act only as aggravating and not as determining causes, that is, abbreviating the latent period.

Having stated this much, and to avoid useless and continual repetitions, I will proceed to succinctly state what are the above mentioned factors, and in what manner their action takes place.

PART I.

FACTORS WHICH EXPLAIN THE CAUSES OF RELAPSES AFTER LONG DURATION, AND WHICH AGGRAVATE RELAPSES AT SHORT INTERVAL.

The influence which good food and sound health has upon the development of malaria in those exposed to malarial infection is remarkable. This is well seen in the case of masters and labourers; for in the case of the former, owing no doubt to better food and environment, relapses and even primary infections are much fewer than in the case of the badly fed and badly housed labourers. It is not altogether the food, however, that plays a part, for in the time of harvest, when the labourer is fairly well fed, relapses are more common amongst the labouring classes than amongst the masters. This no doubt is due to the extra work and consequent fatigue which follows, and to the fact that gastro-intestinal ailments are apt to develop owing to the extra eating and the drinking of wine which the labourer during harvest work partakes of.

Gastro-intestinal troubles are no doubt accountable in many instances for the reappearance of the attack of fever, for it invariably follows that after any rural feast such as takes place in connection with religious ceremonies, weddings, and at the end of harvest, a sudden accession of relapses occurs.

This is also noticed in mountainous, non-malarious districts, and I noticed in a small mountain village a relapse occurring in three young fellows, who about two days before had been at a rural ball and at the subsequent festivities. No other cause could be ascribed for the relapse, yet in one seven months after, and in the other two ten months after primary fever, relapses occurred; it is also worthy of note that these were the only persons in this village who had taken part in the festivities.

It is admitted that after periods of great fatigue relapses are common.

Traumatism would also appear to exercise a determining influence on recurrences of fever. I can recall the case of a patient in my ward who had been ill for several months with cardiac trouble, falling out of bed, and after forty-eight hours developing fever of the quartan type, a type of the disease from which he had previously suffered. In patients, coming from the country to a city hospital, suffering from traumatic ailments, it is quite usual to find that they develop malarial febrile attacks after two or three days in hospital.

Sudden chills and damp would also appear to play a distinct part in causing relapses. Labourers who sleep out of doors or in exposed huts are apt to wake up in the early morning with a feeling of chill, which is but too often followed by an attack of fever. When a group of labourers are caught in the rain I have frequently noticed that after one or two days

those who have had malaria previously are very likely to develop a relapse.

That excessive heat definitely influences the occurrence of relapses I would not venture to say. On the contrary, it is well to observe that, during the three periods of excessive fatigue for the labourers, namely, at the harvest of the grain, of the Turkish corn, and of the olives, under the same conditions of nourishment and fatigue, I found the relapses equally numerous, and in the same proportion, although these three periods fall the first in summer, the second in autumn, and the third in the depth of winter. Labourers who contracted fever whilst working in the plains in the summer and recovered, have on returning to their mountains had relapses in the autumn. Their belief that the mountain air is the cause of the relapse is such that they are ready to leave their homes again, and to go to the plains for the vintage or to sow corn.

One person had caught a tertian spring infection in the summer of 1899, and had numerous relapses in the winter, staying in Amatrice, a mountainous non-malarial place; and this was repeated every time that he went hunting, during which time he spent many hours in the snow often without food. It is quite enough to pass from a warmer to a colder region or the reverse, for the relapse to develop, in fact this is so well known that the labourers believe that the mountain air produces the fever.

Certain foods and medicines are by some stated to favour relapses. This in all probability is due to the gastro-intestinal troubles set up by these substances and not to any specific feature of either the food or medicine. In one case a relapse occurred every time the patient ate fruit, more especially peaches. On careful enquiry, however, it was apparent that the febrile attacks were in every instance attended by vomiting and purging. Another patient got a relapse of fever every time he took iodide of potassium; he took the drug for general arterial sclerosis from which he suffered, but with every administration attacks of tertian fever developed and continued in spite of quinine. In this case also the relapses were associated with gastro-intestinal troubles which were set up by even minimum quantities of the drug.

To intercurrent illnesses are ascribed an influence in the shortening of the period of latency; and it is a question whether the diseases themselves favour fresh relapses or whether they are produced by remedies such as bathing, wet-packs, &c., which form part of the treatment in typhoid fever, &c.

One peculiar feature about relapses in malaria is that they occur in spite of prophylactic treatment by quinine, or by quinine combined with iron and arsenic. At the same time it must be admitted that the period of fever is shorter, the attacks more irregular, and the temperature less high. In fact my observations agree with that of many others, namely, that however potent quinine, &c., is in preventing primary infections, and in the treatment of fever after a primary attack it would seem to have but little influence in chronic malaria. With this conclusion Koch does not agree, for he is of opinion that quinine and methylene blue are valuable prophylactic remedies in all stages of malarial infection.

It would appear that neither age nor sex have pronounced determining influences in malarial relapses. Amongst the very old and the very young the attacks of fever may be fewer from the fact that they are less exposed to such conditions as are calculated to induce malaria, such as cold, fatigue, exposure to great heat whilst at work, &c. In malarial districts, however, where whole families reside, Koch maintains that the children suffer most, and that in their blood the greatest number of parasites will be found, and if careful observations of temperature, &c., are made, that relapses are more common amongst them than amongst any other section of the community.

(To be continued.)

NOTE ON THE INDIANS IN THE CAQUETA TERRITORY, COLOMBIA.

By MONTAGUE D. EDER, B.Sc.Lond., M.R.C.S.,
L.R.C.P.Lond.

THE accompanying photographs were taken in a recent trip to this unexplored region. After crossing the Eastern Andes from Colombia the following rivers were visited: the Orteguasa, Caqueta known in its explored course below the falls as the Jupura, Caquan, and some of their affluents. The tribes inhabiting this district are:—

(1) *Tamas*, on the left bank of the Orteguasa. There are now about forty persons; two years ago nearly eighty died of the small-pox.

(2) *Coreguajes*, on the right bank of the Caqueta; counts some 300 in all. The language differs from *Tama*, but ornamentation and habits very similar. Both these tribelets live in fear of, and cannot be induced to go down the river to, the *Uitote*.

(3) *Uitotes*, who inhabit both banks of the Caqueta at its junction with the Caquan and spread up into some of the near tributaries. The estimate I was given of 20,000, is probably erroneous and made to include a number of distinct tribes. Along the river Yari offshoots of this tribe practise cannibalism. The *Uitotes* are fierce fighters, a decidedly handsome race and less morose than the other tribes. A few of the men wear the "cusma," a chemise-like garment, but all the women and most of the men go naked and unashamed.

All these tribes (men and women) eradicate the hair from the body saving their chevelure; eyebrows, eyelashes, hair from the face, axillæ, genitals, all is pulled out. The reason given me by one hairless genius was they could see the fish in the water more quickly; obviously a very insufficient reason. The *Tamas* and *Coreguajes* (which tribelets I had most opportunity for observing) are nearly all disfigured by blue carate (pinta disease). Though not soap users they bathe at frequent intervals throughout the day; babies of two months or so are ducked in the rivers by their mothers and howl thereat like any highly civilised babe of the same age. Caries of the teeth is very prevalent in these two tribes; they blacken their teeth with some juice, but I cannot say whether this is the cause of the decay. Phthisis and pneumonia not infrequent; small-pox epidemic and very fatal; syphilis apparently unknown, but gonorrhœa occurs.

Malaria exists in the region, but I think these Indians are really immune; no signs of cachexia were noticed in young or old. Among the few half-breed whites one encounters, the Indians enjoy a great reputation for a knowledge of medicinal herbs. It may have been that professional jealousy would not allow them to impart their secrets to me, but I think the doctor cures on modified Christian-science methods. These same "whites" were not loth to be cured by my quinine, calomel, &c. The food is chiefly farinha and cassava from the wild yuca, plus the products of fishing and hunting; monkey is a much esteemed meat. For about six months in the year the tribe has a common dwelling-house; in the summer, when the rivers dry and sandbanks appear, small huts are erected on these banks; each hut inhabited by a separate family. "In the spring a young man's fancy lightly turns to thoughts of love." I was told that it is only at this summer season that the sexes cohabit. Monogamy is the rule and the husband is very solicitous for the well-being of wife and children. The photographs show:—

(1) *Tama* Indians, male and female; note carate on both faces, woman's hands and the man's legs.

(2) *Coreguaje*, epaulettes made from palm leaves; the chest lace is of seeds and various aromatic woods.

(3) *Coreguaje*, note wood through nasal septum; ear doubly pierced for arrow and triangular earring from a battered silver dollar.

(4) *Uitote* boy, good looking. This boy had been brought by a trader, a gift from the parents; he was going to the interior of Colombia as "general" to some merchant.

It may interest anthropologists to note in the photographs that there is nothing very Mongolian about the features. As regards language and general anthropometric data, a whole continent separates the Red Indian from the Yellow race. I believe it is now generally agreed that the American Indian is of a race apart and not a Chinese offshoot, the easy and earlier view.

NEW GROWTHS IN TROPICAL COUNTRIES.

By A. B. DALGETTY, C.M., M.D.
Adampore, South Sylhet, India.

THE geographical distribution of disease, and the question of a probable antagonism between different diseases are subjects of much interest and importance, but they are matters in which great care must be exercised with the premises in order to arrive at correct conclusions. There is an opinion prevailing that neoplasms are comparatively uncommon amongst the inhabitants of tropical countries, and it has even been suggested that the presence of malaria is responsible for this supposed immunity. The matter is very complex, owing to the great number of questions to be considered, and the data, so far, are meagre and inexact. Hence it is that I venture to send you this small contribution to a great subject, with the hope that it may help to forward a knowledge of the matter.

In India, religious and caste prejudices greatly

hinder advances in medicine generally, and not less in this subject of new growths in particular, because the females of the community are hardly allowed to be seen, and it is specially amongst them that one would look for neoplasms.

However, after one's reputation has become established, it is surprising the amount of confidence which the natives will place in him, and the amount of professional freedom which he is allowed to exercise, in spite of the caste prejudices and religious superstitions of the people.

During the past five years I have had close acquaintance with a labouring population numbering about 12,000, comprising men, women and children, who are imported from other parts of India to work in the tea gardens; and during the same time I have come in contact with a great many of the permanent residents of the district who have come to me from time to time with various illnesses. These were drawn from a population numbering at least another 15,000, so that in these circumstances one should have gained an experience at least approximately accurate of the different kinds and relative frequency of the endemic diseases.

The imported coolies are all Hindus, the resident population mostly Mussulmans, born and bred in the district for many generations.

During the same period I have had under my care thirty-seven Europeans consisting mostly of men from 25 to 40 years of age, but including also five women and two children. The following table gives the result of my experience of new growths, innocent and malignant, during these five years.

The division into Hindu and Mussulman serves both to distinguish the races and to separate the list of imported coolies, concerning whom my knowledge is much more accurate, from the residents of the district concerning whom my knowledge is less accurate.

MALIGNANT TUMOURS.

| No. | Race. | Sex | Age | Growth | Site |
|-----|-----------|-----|-----|-------------------|----------------|
| 1 | European | F. | 47 | Cancer | Breast. |
| 2 | | M. | 35 | " | Tongue. |
| 3 | | M. | 45 | " | Lip. |
| 4 | | M. | 35 | " | Gum and cheek. |
| 5 | Hindu | M. | 30 | " | Glans penis. |
| 6 | | F. | 40 | " | Os uteri. |
| 7 | | F. | 40 | " | " |
| 8 | | M. | 35 | Sarcoma | Palmar fascia. |
| 9 | | M. | 35 | " | Rib. |
| 10 | | M. | 50 | Cancer | Lip. |
| 11 | Mussulman | M. | 35 | Sarcoma | Scalp. |
| 12 | | M. | 30 | Melanotic sarcoma | Eye. |

NON-MALIGNANT TUMOURS.

| | | | | | |
|----|-----------|----|----|----------------|----------------|
| 13 | | F. | 40 | Ovarian cyst | — |
| 14 | | F. | 30 | " | — |
| 15 | Hindu | F. | 35 | Fibroid | Intra-uterine. |
| 16 | | F. | 25 | Lipoma | Breast. |
| 17 | Mussulman | F. | 45 | Cystic fibroid | Extra-uterine. |

* The ages of the natives are only approximately correct.

Several other cases of suspected malignant disease of the intestinal tract have been seen, but owing to the difficulty of getting *post-mortem* examinations the diagnosis remained unverified. The European case had lived for twenty-five years in India, mostly in this district. The breast, axillary glands, and a large part of the pectoral muscle were removed by Professor Ogston, of Aberdeen, and at this date, four years after, there has been no recurrence.

The figures given for the Hindu population represent fairly accurately, I think, the actual state of matters as regards new growths amongst them, with this exception, that I suspect there are some cases of uterine disease which are hidden. These figures show only eight cases of malignant disease within a period of five years in a population of 12,000 of all ages.

The sites of growth given above cover the most part of the field usually occupied by the malignant neoplasms, with one notable exception, namely, the female breast.

It is a remarkable thing, but it is a fact that I have not yet seen a case of malignant disease of the mamma in a native of this country. Why this should be is a mystery. It can hardly be simply that I have been unfortunate in this respect, for cancer of the breast would be difficult to hide in the open dress of the native, and other diseases of that part incident on lactation, want of cleanliness and the like are frequently seen.

One or two points in the habits of the natives of India contrast sharply with those of Western races. In the first place there is dress. A woman's dress consists simply of a cotton cloth passed round the body and pulled up over the head and shoulders so that no pressure is exerted on the breasts, which hang loosely and freely.

In the second place, there is the custom of oiling the body. The oil used here is chiefly from the cocoa-nut and every man and woman, even the poorest, tries to get a little of this substance, which seems to be a necessity of life.

It is rubbed into the hair and all over the body and appears to check too rapid evaporation, to protect the skin from over drying and blistering from the sun and to cause the rain to run off as from the feathers of a duck. It may be mentioned incidentally, that ringworm of the scalp is extremely rare here, although ringworm of the body is very common and it is just possible that this free oiling of the hair has something to do with its prevention. The oil sticks for a much longer time amidst the hair than on the surface of the body.

In the third place, mothers suckle their children much longer than is the custom in the West. They cannot afford to buy much milk and the same reason saves them from the deluge of artificial preparations which flood the markets at home; besides, the mothers do not look upon the nursing of their own children as a hardship and an unjust task of Nature to be avoided if possible and certainly to be curtailed to the barest necessity. Much the reverse; the mother is the slave of the child and willingly yields her utmost service. The child comes backwards and forwards to the breast long after it is able to speak and run about. This continues for a year, fifteen months, eighteen

months or even longer, so that the lately formed cells of the gland undergo a gradual and complete devolution instead of their function being suddenly checked while they are still in full activity. The breast is literally sucked dry. Such a gland would appear less liable to undergo perverted action than a gland arrested while its function is still in full force.

To digress again for a moment, may it not be that this prolonged suckling accounts in some measure for the excellence of the teeth in the mouths of the less civilised races.

Of course, the three points mentioned above, if they have any influence at all, can only be factors contributory towards the repression of the causes supposed to favour cancer, the true cause being, as we must regard it to be, a living organism.

Again, it may be said that **Eastern races die at an age when cancerous growths are less common.** But age is a relative term. In the tropics the succeeding stages of life follow one another quickly. There is properly hardly any period of youth. Children shoot up from childhood into manhood almost at a step. Amongst the Hindus, marriage is arranged for the children while they are still mere infants and when sexual maturity arrives they begin life together, hence by the time they are twenty years of age they are the parents of a considerable family. Old age, therefore, comes quickly, especially to the labouring classes; a man is old at thirty and not fit for much at forty-five, if he survives till then. Thus the Western cancer-age is hardly reached although one should expect that it would be merely anticipated by ten or fifteen years if the disease were as common in the East as it is in the West.

Another matter worthy of mention is the **absence of ordinary warts.** I do not remember ever having seen a wart, a thing which is so common amongst people at home, on the hands or any other part of the body, either of a child or an adult in this country. Why this should be is difficult to say.

Granting for the sake of argument that new-growths are less common amongst less civilised races, has the presence of malaria anything to do with it? In tropical countries malaria is ubiquitous and everyone suffers from it more or less at some period of life. It is safe to say that everyone of the cases in the list given above had suffered from malaria, so that the two diseases are not absolutely antagonistic at any rate. If one could find a region where malaria was abundant and cancerous diseases almost unknown and a contiguous region where the state of things was exactly the reverse, something might be said for a possible antagonism; but such a contrast would be very difficult to find. Or again, in an individual suffering from undoubted cancer, if the onset of a severe malarial fever led at once to a marked diminution of the growth there would be good grounds for connecting the two as cause and effect, but I have seen no case of the kind.

However, the subject is a new one and time will be required to collect data.

BLACKWATER FEVER.—M. Otto reports a case of blackwater fever occurring in quartan malaria as illustrative of the theory that quinine is the causative factor of hæmoglobinuria in malaria. In this case the blood pigment first made its appearance after the exhibition of 75 grains of quinine. The patient had been suffering from malaria for some weeks, but quinine had not been previously taken except in one of two very small doses. The hæmoglobinuria was very pronounced, but no more quinine being given, soon subsided. In a few days quinine was again administered, and was promptly followed by the presence of blood pigment in the urine, but this second attack was not as severe as the first. The administration of the drug was then carried on by the rectum and was not followed by hæmoglobinuria; it is probable that this was due to the small amount thus absorbed. Later renewed attempts were made to give quinine by the stomach, but whenever any considerable amount was taken hæmoglobinuria promptly occurred. The vast majority of the cases of blackwater fever hitherto observed have occurred in tropical countries, and in connection with the pernicious form of malaria. The fact that this case developed in Germany, in a patient who had never been outside the limits of the country, and was plainly brought on by the administration of large doses of quinine, lends strength to the theory that this disease results from the action of quinine upon the blood of certain malarial patients. — *Deutsche Medicinische Wochenschrift*, January 23rd, 1902.

A NEW FACTOR IN THE ETIOLOGY OF MALARIAL FEVER, INDICATING NEW METHODS OF TREATMENT.—A. F. A. King adduces arguments to prove that heat is not a factor in the etiology of malarial disease, but explains the undeniable relation between hot climates and malarial fever by eliminating the term "heat" and substituting that of "light." Paroxysms of intermittent fever will not, as a rule, take place at night, in the dark. In places where malarial fever prevails, the disease is increased by bright, sunny weather, and lessened by clouded skies. It has long been a tradition that to prevent the occurrence or recurrence of ague it is advisable to keep in the shade and avoid sunlight. The malarial parasite is a naked amœba. Red light promotes the vital activities of amœbæ, while violet or purple light restricts them. The colour of the light diffused through the blood is necessarily red. The relative liability and immunity of different races of men to malarial fever depend upon the relative translucency or non-translucency of their skin, and probably of their blood. If the etiology given be correct, correct treatment will consist in keeping patients in the dark, or in rooms with purple or indigo windows, and clothing them in garments impenetrable to light; in the tropics, white clothing, lined with purple or black. Drugs that darken the blood, or render it violet, or lessen its translucency, should be given. Quinine sulphate in solution intensifies the violet, and even renders the ultra-violet rays of the spectrum perceptible to human vision. — *American Journal of Medical Science*, February 10th, 1902.



FIG. 1.



FIG. 2.



FIG. 3.

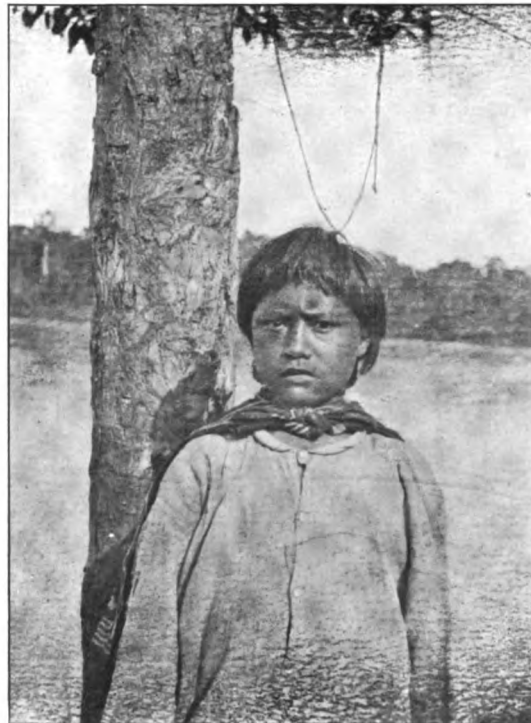


FIG. 4.

To illustrate Dr. EDER's paper—"Note on the Indians in the Caqueta Territory, Colombia."
(See page 122).

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THE

Journal of Tropical Medicine

APRIL 15, 1902.

NEW GROWTHS IN TROPICAL COUNTRIES.

DR. DALGETTY'S paper in the present issue of the Journal is opportune. It moreover conveys a considerable amount of useful information, and, at the same time, introduces a number of original ideas well worthy of discussion and close attention.

The relation of malaria to cancer, so widely published at the present moment, must cause every practitioner in the tropics to think over his experiences, and to collate for publication or for his own knowledge, the results of his observations. Many will no doubt deride the idea, and others may uphold the relationship of the two diseases ; but all would do well to be warned by Dr. Dalgetty's remarks, that the information is not so easily obtainable as would at first sight appear. We know how many women in Britain seek to conceal the fact from their friends and from their doctors that they have any growth in the mamma.

We have even heard it said that should a woman come to consult a surgeon at an early period of the growth of a mammary tumour, it affords a presumption that the disease is not malignant. It would seem to be a feature of the constitutional or mental condition of women suffering from malignant growths that they wish to conceal their ailment. An explanation of this is difficult to find, except it be the dread of having the suspicion confirmed, or of the surgical proceedings which may be recommended. In tropical countries there are several other reasons ; there is the dread of the foreign doctor, the seclusion in which women naturally live, the reticence to bring any feminine ailment within the ken of the male, and the horror with which native women regard operations ; medical men therefore collecting statistics concerning diseases of natives, more especially of the nature of new growths, have to exercise the greatest care that they are not misled. How often has it happened that, after years of residence in any locality, certain types of disease seem to be non-existent until some chance circumstance brings one case forwards. Should this be of the nature of a tumour, and if the surgeon is fortunately able to successfully remove the growth, many more cases will immediately come forward for treatment. Take such an ailment as ovarian tumour. The writer has previously related his experiences in this direction : how for four years no case of ovarian tumour was seen by him amongst native Chinese women, and yet, after a successful operation on one patient, a large number of women, suffering from ovarian tumours, immediately came forward for treatment. When cholera or plague is reported in a native city, a doctor sent thither always finds considerable difficulty in getting to know of any cases, and establishing the fact that the report is true. Cholera and plague cases are carefully hidden when an inspection is being made, but once a few cases are found, and the natives see that the patients are kindly treated, the inspecting officer may suddenly find himself in the midst of a severe epidemic. If this is the case with an epidemic disease, how much more likely are cases of malignant disease capable of being concealed ?

Lactation and Cancer.—Dr. Dalgetty's remarks on the period of lactation in women and on the emptying of the mammary gland are well worthy of study. The native women suckle their children until the gland "runs dry," and Dr. Dalgetty not inaptly ascribes the absence of mammary cancer to this circumstance. The pernicious doctrine—taught chiefly by the monthly nurses who attend upon "fashionable" women in Britain—that the child should be reared by hand, and that the mother is "not strong enough" to bring up her child at the breast, is at the root of many of the troubles of both women and their children. Measures, abnormal measures, are taken to check the flow of milk, and the normal physiological function of the gland is arrested by abnormal means. That unhealthy processes must ensue would seem physiologically certain, and it may be that we have here one explanation of the frequent presence of mammary cancer amongst the women of Western Europe compared with the same disease amongst natives of the tropics. We are aware, of course, that barren women are attacked by mammary cancer, but in them it is usually at a much later period of life.

Dr. Dalgetty's remarks on the teeth of native children are also worthy of note. Decayed teeth in young people of the tropics is quite the exception; until the age at which tobacco is used, or some one of the baneful customs, such as teeth-staining, betel chewing, &c., are in vogue, carious teeth are seldom seen. Another point connected with early weaning from the breast is the fact that the mother becomes again pregnant soon after the birth of a child. As long as lactation continues she is not so likely to become pregnant, and the intervals between her children are therefore two or three years as a minimum. In the countries of "higher" civilisation, in which the period of suckling is curtailed, the mother may become pregnant within a few months of parturition, and it may happen that five or six children are born in as many years. That this can be good for either mother or children is unlikely, and with it the modern plan of "prevention" came into vogue. The matter resolves itself into a simple problem: Allow the natural period of lactation to

continue and the intervals between the birth of children will be two or three years; curtail it by artificial means and either one of two things will happen: the mother will have her children too quickly or she will resort to artificial means to lessen the number of her offspring. The lesson for medical men is clear, namely, to encourage the mother to bring up her children by the breast for at least nine months, and to set their faces against the nurses who, pandering to the wishes of their employers, recommend the pernicious and unhealthy doctrine that the child is best reared by "the bottle."

J. C.

MEDICAL SOCIETY.

MADRAS BRANCH, B.M.A.

THE following interesting and valuable discussion is published *in extenso*.—

Discussion on the Treatment of Hepatic Abscess.

LIEUTENANT-COLONEL J. MAITLAND, I.M.S.: The subject that we have met to discuss this evening is not only one of perennial interest to us in Madras, where the disease is so common, but is also one of special interest at the present time, owing to the fact that somewhat radical changes in the treatment of this affection have lately been forced upon the attention of the profession. The first point to which I should like to draw attention concerns the use of the exploratory needle. There is a tendency amongst medical men of the present age to resort somewhat hastily to the use of such means as that of the exploratory needle, to the neglect of careful and systematic investigation by the more ordinary and often less hazardous methods of clinical examination—a tendency, in other words, to make short cuts to diagnosis. I am afraid that this is sometimes the case in regard to abscess of the liver. In the great majority of cases the diagnosis of hepatic abscess is quite simple, and the use of the exploratory needle quite unnecessary. It is extremely important to emphasise this point because there appears to be a widespread idea that the use of the exploratory needle, in cases of disease of the liver, is a very simple procedure, and one unattended with any danger. So far is this from being the case, that a very considerable number of deaths have been recorded, as having been due directly to this operation. For this reason alone it is important to insist that exploratory punctures for the purpose of diagnosis in cases of disease of the liver should be restricted to those cases in which it is absolutely necessary. There are a certain number of cases in which its use cannot be avoided, but they form a comparatively small proportion. What are the causes of the hæmorrhage which occurs in some of these cases? Mr. Cantlie, in a paper read at the recent meeting of the British Medical Association, stated his belief that the chief danger of hæmorrhage was from the inferior vena cava, and that this vessel could not be injured so long as the needle was not thrust in as far as four inches. Whether any of the recorded cases of hæmorrhage were due to injury of the vena cava or not, I am not prepared to say, but I do know from practical experience that fatal hæmorrhage may occur from puncture of the liver tissue alone, when that tissue is in a condition of acute congestion. I speak feelingly on this subject because I lost a patient myself

from this cause. The case was one in which I was asked to explore the liver of a patient who was suffering from acute hepatic congestion, and in which the presence of an abscess was suspected. Two hours later, when going round the wards, I found the patient dying from internal hæmorrhage. The vena cava was not injured in this case; bleeding took place from the liver substance itself. Fatal hæmorrhage has also been known to occur from injury to the intercostal artery. Whenever it becomes necessary to perform an exploratory operation, arrangements should be made to open the abscess at once, should pus be discovered. By opening the abscess at once, not only is the patient saved the injurious consequences that may result from delay, but he is also saved the anxiety involved in a second operation. Moreover it sometimes happens that considerable difficulty is experienced in hitting off the abscess at the second operation, although it may have been found quite easily on the first occasion. Indeed I have known of a case, in the practice of a colleague, in which the abscess could not be found at all at the second operation. In performing the exploratory operation, by means of a needle there is one precaution to which it is important to pay attention. There is always a temptation, in withdrawing the needle, when no pus has been found, to alter its direction before it has been completely withdrawn, and make another exploratory venture through the same orifice. As this manœuvre tends to enlarge the orifice of entrance, it should always be avoided. If it is necessary to explore again, a fresh puncture should be made at another point. The fatality to which I have already alluded was due, I believe, in part, to neglect of this precaution. The possibility of wounding the intercostal artery, if due precaution is not taken, must also be borne in mind.

I will now pass to the consideration of the operation for the evacuation of the abscess. If no exploratory puncture has previously been made, a needle must first be inserted for the purpose of locating the pus. In the great majority of cases of single abscess the pus is found in the right lobe, and a trans-thoracic opening is required for its evacuation. The cases in which the abscess is situated in the lower part of the right lobe, which has been pushed far down below the margin of the ribs, are, in my experience, comparatively uncommon. I propose, therefore, to first describe the trans-thoracic operation, as I am in the habit of performing it. If any point of special tenderness has been found, the needle may be thrust in at that part. If no indication of that nature is present, it is best to explore through the eighth intercostal space. When the abscess has been found the needle should be left *in situ*, to serve as a guide. An incision is then made over the rib immediately beneath the level of the needle, and a portion of the bone, about two inches in length, is removed. The pleura is next incised, and, if there are no adhesions, the two layers are stitched together, the sutures being made to enclose as wide an area as possible. The diaphragm and peritoneum are next divided, and if no adhesions are present, the peritoneum is dealt with in the same manner as the pleura. A pair of sinus forceps is passed alongside of the aspirator needle into the abscess and an opening made by separating the blades. The opening is then enlarged to the necessary extent by incision. Having made a sufficiently large opening, two glass drainage tubes are inserted and the pus allowed to flow away. When pus has ceased to flow freely the cavity is irrigated with weak boric acid lotion or sterilised water, in order to wash away as much as possible of the remaining purulent contents. Irrigation of the abscess cavity, although not an absolute essential, has the advantage of minimising the amount of discharge subsequent to operation. After the abscess has been emptied as far as possible, a finger is introduced into the cavity for the purpose of ascertaining its size, and necessity, or otherwise, of making a counter-opening. If the abscess is a large one, and the opening has not been made at the most dependent point, a counter-opening should be made further down and more

posteriorly. The arrangements for securing free drainage are the most important points in the whole operation, and it is for this reason chiefly, as I shall show later on, that I am opposed to the methods recommended by Mr. Cantlie, in the paper alluded to above. The main opening into the abscess should be sufficiently large to admit two drainage tubes of half an inch diameter easily. An opening of this size cannot be made in the chest wall without removing a piece of rib. I am in the habit usually of injecting iodoform emulsion into the cavity of the abscess, before inserting the drainage tubes. This is done with the view of checking septic decomposition should the discharges come to the surface and become contaminated, as they are not unlikely to do if the abscess has been a large one. There is considerable difficulty in applying a sufficiently wide and thick dressing to this part of the body. In a climate like that of this country, nothing is more distressing to a patient than to be swathed in an enormous mass of dressings.

When the abscess has to be opened below the ribs the operation is necessarily more simple. Here again I consider it best to operate by means of a free incision, and, if adhesions have not taken place, to stitch together the two peritoneal surfaces. If, as sometimes happens, difficulty is experienced in suturing the peritoneum, owing to the proximity of the pus to the surface, or to the movements of the liver, the better plan is to pack the wound with gauze, and, having turned the patient on his side, to open the abscess by means of a knife. After the pus has been evacuated, the edges of the wound in the liver are stitched to the edges of the wound in the abdominal wall.

I should now like to say a few words regarding the method of operation recommended originally by Dr. Manson and specially advocated by Mr. Cantlie at the recent meeting of the British Medical Association. It is necessary that this question should be fully discussed, not only because this method is so enthusiastically recommended by Mr. Cantlie, but also because he condemns, in equally forcible language, all other methods of operation. Is it possible that our methods here in India are so faulty as he proclaims them to be? For my own part I do not think so. Mr. Cantlie's method, speaking briefly, consists in puncturing the abscess with a trochar and emptying it by means of "syphon drainage." It is in fact an operation by means of limited incision as opposed to operation by free incision, and is therefore opposed to the ordinary principles upon which abscesses in other parts of the body are treated. The main objection to this operation, as I have already stated, is that by such a method of dealing with an abscess of the liver we cannot ensure that drainage will be sufficiently free. This objection is forcibly illustrated by three out of the four cases cited by Mr. Cantlie in his paper. In the first case we are told that on the tenth day the drainage tube having slipped out, it was found necessary to administer an anæsthetic before the tube could be re-inserted. Again, in the same case, on the thirty-fifth day, it became necessary to make a counter-opening in order to establish free drainage. In the second case we find that on the thirty-third day there was some difficulty in drainage, and it was contemplated to open the cavity further back, but the patient objected to be operated upon again. In the third case the drainage tube was pulled out by the patient on the night after the operation and "could not be satisfactorily replaced." Four days later a large drainage tube was introduced and twenty ounces of pus welled out of the wound. It is to be presumed that these four cases that have been selected by Mr. Cantlie, are taken by him to illustrate the benefits of his method of treating abscess of the liver, as compared with the results obtained by other methods. If that be so, I can only say that in my experience such extraordinary difficulties in securing free drainage as were met with in these three cases are the rarest exception. It is impossible to avoid the conclusion that all these difficulties would have been obviated had the abscesses been opened by free incisions. The truth of the matter is that an opening so small as to

tightly embrace a single tube does not suffice for the efficient drainage of most large abscesses of the liver. Moreover, if the tube becomes displaced by accident, its replacement into so small an opening is almost an impossibility. These difficulties are occasionally met with even in cases where a piece of rib has been excised and an opening made sufficiently large to admit two drainage tubes with ease. Another disadvantage of Mr. Cantlie's method of operation is that it does not admit of exploration of the cavity of the abscess with the finger, and therefore no estimate can be formed of its size, nor of the necessity, or otherwise, of making a counter-opening.

Before concluding I would like to draw attention briefly to the objections raised by Mr. Cantlie against the ordinary method of operation. In the first place, he states, that "the severity of the operation in many cases is such that the opening of the abscess is apt to be deferred until too late in the disease." That is a statement that is entirely without justification so far as India is concerned. The rule in this country is to operate at once. His second objection is that "the medical practitioner is willing to try every available resource before condemning his patient to so severe a line of treatment." It is hardly necessary for me to say, in this assembly, that such a statement is also without justification, at least so far as India is concerned. Indeed it would be curious to know what the "available resources" are, to which Mr. Cantlie alludes.

LIEUTENANT-COLONEL STURMER, I.M.S.: I agree with what Colonel Maitland has said for the most part. My experience is no doubt limited and I have not had to excise the rib so frequently, but then perhaps my cases have been further advanced and have contracted adhesions to the abdominal wall below the ribs. I cannot consider Mr. Cantlie's operation superior to that of incision and free drainage. My results from prospecting the liver have never resulted in any after bad effects, but Colonel Maitland states he lost a case, and Colonel Hatch, in the *Indian Medical Gazette*, reports one or two deaths. That the liver can bleed when punctured there can be no doubt, and that too, without puncturing any such large vessel as the vena cava. I certainly think it advisable to irrigate the abscess cavity after incision, and cannot understand why you should treat a case of hepatic abscess differently to that of an abscess elsewhere.

CAPTAIN MOLESWORTH said he should like to ask Colonel Maitland whether it was not a fact that exploration in cases of congestion of the liver has not been followed by disappearance of the symptoms.

LIEUTENANT-COLONEL BROWNE agreed with Colonel Maitland's remarks. He did not quite understand what Mr. Cantlie meant by "syphon drainage," and noticed that Dr. Manson said nothing about it in his latest book. It was very unusual for the tube to run "full," and therefore "syphon action" could not be obtained. He believed that the danger of damaging the pleura was just as great with the tube as with the incision method. He knew of three cases in which death occurred from hæmorrhage. In one of these, certainly, the bleeding did not come from the vena cava or any of the large veins, but welled up from the whole of the interior of the wound.

CAPTAIN NIBLOCK said that he agreed with everything Colonel Maitland had said. As to exploratory puncture with a needle he thought it should only be done in the operating theatre, when the operation could be proceeded with at once. Two cases he had seen impressed this strongly on his mind. The first case had been explored in the ward, and was a short time afterwards sent to the theatre. When the liver had been exposed by incision the pus was seen to be pouring into the peritoneal cavity through the puncture in the hepatic wall. In the second case also the patient had been punctured and then sent to the operating theatre with diagnosis of hepatic abscess. Under chloroform an exploring needle was introduced close to the former puncture wound, but immediately on entering

the cavity of the peritoneum blood began to flow through the needle and several ounces came away. An incision was made alongside the needle at once, when about ten ounces of dark blood were sponged out of the peritoneal cavity. The liver was found to be very congested and the capsule extremely tense. An irregular rupture about three-quarters of an inch in length was found in the latter, which opened immediately into a cavity the size of a walnut. In this case the oozing was general and did not come from any large vein. The damage to the liver substance was probably due to alterations made in the direction of the needle before it had been completely withdrawn. His experience therefore agreed with that of the previous speakers that bleeding was not in every case, at any rate, due to puncture of a large vein.

The operation performed by him was similar to that described by Colonel J. Maitland, except that he did not inject iodoform emulsion. Later, if any unpleasant smell was noticed in the discharge, he injected iodoform emulsion. He believed firmly in the use of two large tubes, one of which could be removed after a few days' time in the majority of cases. In one of his earliest cases in which the liver only projected a few inches below the costal margin he incised below the ribs, with the result that as the abscess began to empty and contract, the liver was drawn up under the ribs and the utmost difficulty was experienced in getting, and keeping, the drainage tubes in. Ever since, unless there was very great enlargement below the costal margin, he used the trans-thoracic method, generally with excision of a rib.

He had operated by the incision method in twelve cases of single abscess, with two deaths, and in four cases of multiple abscess, all of whom died. In no case was death due to the operation itself, and he did not think that any of the fatal cases would have been saved by the "syphon drainage" method.

LIEUTENANT-COLONEL J. MAITLAND in reply: Captain Molesworth has drawn attention to the fact that it is stated in some books that puncture of the liver with an exploring needle, even if no pus is found, is calculated to have a beneficial effect. It is unfortunately true that such has been the teaching on this subject, but it is to be hoped that, in view of the fatalities that have resulted from the operation, the use of the needle will in future be more restricted than in the past. He was pleased to learn that evidently all the members present were in favour of the open method. As to mortality after operation, the great majority of our deaths were in cases of multiple abscesses of the liver.—*The Indian Medical Gazette*.

New Drugs, &c.

SINARO WATER.

A TABLE WATER.

THE market is so flooded with mineral waters of various kinds that it seems waste of space to discuss another. After a practical test, however, of Sinaro, we have departed from our plan of ignoring such waters and the flaming advertisements of their promoters. Sinaro is not only a pleasant water for table purposes, but we can positively assert its efficacy in kidney complaints of the nature of gravel. The composition of the water no doubt helps to explain its efficacy in complaints of the kind, but practice is worth any amount of theory, and the practical value of Sinaro we are prepared to stand by.

As a test of the usefulness of this natural spring

water in the tropics, we are informed that Sinaro is largely exported to South Africa, Australia, Sumatra, &c.

Analysis of Professor Fresenius, Wiesbaden.

| | | |
|----------------------------|-----|----------|
| Bicarbonite of soda ... | ... | 0.641067 |
| " " calcium ... | ... | 1.207257 |
| " " magnesia ... | ... | 0.430060 |
| " " iron ... | ... | 0.097792 |
| Sulphate of potash ... | ... | 0.008334 |
| Chloride of potash ... | ... | 0.009706 |
| " " sodium ... | ... | 0.001275 |
| Silicic acid ... | ... | 0.035201 |
| Free carbonic acid gas ... | ... | 2.678752 |

Gr. 5.109444

The springs from which Sinaro is derived are situated at Nastaetten, near Wiesbaden. The proprietors of Sinaro have not as yet advertised the water widely enough to make it generally known, but we would advise them to do so, for they have a capital article to push, and it is their fault if the water does not become popular, for it only wants to be known to supersede many at present in the market.

News and Notes.

CHOLERA.—The present pilgrimage to Mecca seems to be attended by a serious outbreak of cholera. Over 1,000 cases are reported to be occurring weekly in Mecca and the port of Jiddah. From Medina also, lying 248 miles to the north of Mecca, over 100 deaths from cholera are reported. Seeing that a large percentage of the Mecca pilgrims go to Medina, the danger of a widespread infection is considerable.

The danger to Egypt from cholera is particularly great. This year a large number of pilgrims have gone from Egypt, and it is to be feared when they return from Arabia that an outbreak of cholera may occur. The Sanitary Department of Egypt is, however, so well regulated, and the authorities are so constantly on the alert, that they may be implicitly trusted to take the necessary steps to guard against the disease gaining a serious hold on the country.

NATIVE REMEDIES.—The *British and Colonial Druggist*, February 21st and of March 14th, 1902, gives some interesting details of primitive methods of treatment.

I.

BOER REMEDIES.—A visitor to one of the camps was surprised to see a cat running about one of the tents with all its fur clipped off. He inquired the cause, and was informed that the fur had been cut off and roasted, and then applied to his child's chest as a remedy for bronchitis. On another occasion a doctor discovered that the parents had killed a goat and cut it open, removing the internal organs. They had then put the child inside, with its head protruding through the opening made by removing the breast bone.

A favourite remedy for jaundice is to rub the

patient's body with cabbage seeds. The seeds are then sown. When they come up the jaundice disappears. There is nothing entirely unparalleled in any of these modes of treatment. The idea of the cabbage seeds is not unlike the old English way of curing the whooping cough by interring it, the patient coughing into a hole dug in the soil; or of rubbing warts with a match stick, then burying the match and waiting for a tree to grow and the wart to disappear! A curious cure for toothache is mentioned. The finger- and toe-nails of the sufferer are cut off short. The parings are put into a bottle with a lock of his hair and some water. The bottle is then corked and buried and the toothache disappears.

II.

TONQUIN AND THERAPEUTICS.—Dr. Violet, a surgeon in the French Navy, has just published some particulars of the stock-in-trade of the native drug dealer of Tonquin. Dr. Violet tells us that the soil itself is the most popular and infallible of all the "drugs" in these regions. Mother Earth is prepared for medicinal purposes as follows: Small mud-pies are made and cut up into small squares; these are flattened out into tablet form and cut into little rectangular pieces. When dried, these pellets are ready for use. The soil chosen is determined by the nature of the disease under treatment; thus, dirt dug up in the vicinity of the doorstep is indicated in cases of abscesses, and useful in painful confinements. Earth freshly scraped up by rats cures paralysis and stops the weeping of children. Thus it must not be thought that the Tonquinese treat all diseases from the same bottle, as it were. One small plot of land may comprise a dispensary complete with fifty different kinds of earth, and the skilled doctor knows just where to dig to find the particular medicine his patient requires.

As in all the old pharmacopœias, the excrements of animals furnish some of the most largely-used remedies. This is the receipt for a cure for small-pox. On the eve of the ninth day of the ninth lunar month a pig, a dog, and a cat are shut up in the same enclosure. For ten days they are fed on rice, and at the end of that period the excrement is collected. This is preserved until the eighth day of the twelfth lunar month—the animals being carefully shut off from the rest of the world during the intervening period—at which date, before sunrise, the matter collected is cooked, and the residue in the crucible is put into a bottle. The maximum dose of this medication is 15 grains four times a day. Vaccination is, however, beginning to become fashionable, and this concoction will probably soon be obsolete. Although these medicines would appear of a somewhat primeval nature, it must be borne in mind that the medical profession is not open to all and sundry; on the contrary, the regulations governing the practice of therapeutics are very stringent. In Annam, before a doctor can practice, he must have studied the medical art for a period of no less than ten years. Not that the diseases are more numerous than in other countries, nor their diagnosis more difficult, but the names and the nature of the innumerable drugs make a long course of study necessary.—*Brit. and Col. Druggist*, March 14th, 1902.

Current Literature.

DR. HEM CHANDRA has contributed a valuable article entitled "The Therapeutics of Semi-Carpus Anacardium" (Dhobi's Nut), to *The Indian Medical Gazette*, March, 1902. The conclusions the author arrives at from practical use of the semi-carpus anacardium are as follows:—

"This is a very reliable drug in the treatment of nervous disorders. The precautions of using milk and ghee must not be forgotten. If this drug be used extensively by scientific practitioners, I am sure they will recommend its introduction into the B. P. Many poor people hold a *mela* once a year. During this ceremony they take its decoction with milk, ghee and honey or sugar. This keeps them free from any disease. During winter consumers of this drug can safely sleep in the open fields without warm clothes. I have been using this drug for more than six years without seeing any bad effect other than erythematous rash. In the Campbell Hospital I have made many bed-ridden cases of disseminated sclerosis walk about in the hospital compound. As an alternative it is very helpful in secondary and tertiary stages of syphilis."

"I have used it successfully in two cases of epidemic dropsy of the legs recently."

NOTES ON THE PREVALENCE OF FILARIASIS IN THE CALCUTTA POLICE FORCE.

By C. R. M. GREEN, F.R.C.S.Eng., D.P.H.Camb.,
Major I.M.S.

Superintendent Campbell Medical School, and Police Surgeon, Calcutta.

DURING the months of October and November last year, I examined the blood of 100 constables newly admitted into the Police Hospital for all kinds of disease. The blood was taken at 10 p.m., and in most cases only one cover-glass was examined.

Prevalence.—I found filariæ in 7 per cent. of the cases. The number of constables affected with filariasis was, however, probably much greater, for one case was examined on nineteen nights (besides being examined several times by day) and a filaria only found on one night, and in another case filariæ were only found on two out of seven nights, and seeing that, only one specimen of the blood was examined in most cases.

Species and Periodicity.—The filariæ had all the characters of the filaria nocturna, the hæmatozoal embryo of the *Filaria Bancrofti*, as described by Manson. The covering and uncovering of the head and the shooting out of a spike or fang, as Manson calls it, from the head being easily seen and interesting to watch in dying specimens.

The members of the Expedition to Nigeria consider the *F. nocturna* and *F. diurna* to be one and the same species, the prevalence of the later by day being due to the nocturnal habits of certain West African tribes. As bearing on this point, I may say that the Calcutta police constable has a good deal of night duty, and both he and his ancestors always took a midday siesta when they could get it. In fact most natives of India are, to a certain extent, nocturnal in their habits.

In these seven cases I examined the blood by day, but did not find any filaria, e.g., one case was examined on—

| | | | |
|-----------------|---------|--------|---------------|
| October 25th at | 8 a.m. | Result | <i>nil.</i> |
| " 27th " | noon | " | <i>nil.</i> |
| " 31st " | 3 p.m. | " | <i>nil.</i> |
| " 23rd " | 4 p.m. | " | one filaria. |
| " 22nd " | 10 p.m. | " | two filariæ. |
| " 31st " | 10 p.m. | " | nine filariæ. |

Dr. Manson, in his article on filarial disease in Davidson's "Hygiene and Diseases of Warm Climates," states that, "although when seen alive in the blood, the embryo *F. diurna* resembles so closely *F. nocturna* as to be practically indistinguishable therefrom, a singular difference is observable between the species when seen *post mortem* on dried and stained slides of thickest blood films." This is that the *F. nocturna* is arranged in graceful curves, while the *F. diurna* looks shrunken and thickened and has assumed a stiff, rigid, ungraceful attitude. Dr. Manson says, "I consider it a diagnostic mark of value."

In the Police Hospital, I observed dead forms of these filariæ—certainly *F. nocturna*—in very stiff, ungraceful attitudes, and exactly like those figured by Dr. Manson as belonging to dead *F. diurna*.

Diseases of the cases in which filariæ were found.—Filariae were found in two cases of dyspepsia, one of incised wound, in one each of bronchitis, dysentery, bubo and syphilis (tertiary).

Amongst those examined were two cases of lymph scrotum with elephantoid fever, but filariæ were not found.

Hydrocele and Filariasis.—The blood in five cases of hydrocele was also examined for filaria with negative results. Hydrocele is a common disease of native constables. That it is prevalent in the districts also from which they come is shown by the fact that out of 631 recruits examined by me in the last six months of 1901, 36.6 per cent. were rejected. Hydrocele was responsible for 27.27 per cent. of the rejections, or more than any other two causes put together. The 631 recruits were affected with hydrocele to the extent of 9.98 per cent.¹

Geographical Distribution.—There are no grounds for supposing that the filariasis with which these men were affected was contracted in Calcutta; in fact one affected man had only recently been recruited. The districts from which those affected came were the following:

| | | |
|-----------------|-----|---------------------------------|
| Oude | { | District of Sultanpur, 3 cases. |
| | | Gonda 1 case. |
| N.-W. Provinces | ... | Ghazipur 1 " |
| | | Arrah 1 " |
| Northern Bengal | { | Gya 1 " |

Total 7

The area of the recruiting ground of the Calcutta Police is fairly shown by the above-named districts. It is desirable that some observations should be made

¹ [An examination of over 1,000 Bihar prisoners in Bhagalpore Jail showed an 8 per cent. prevalence of hydrocele.—Ed. I.M.G.]

as to the prevalence of filariasis in the inhabitants of Lower Bengal.—*The Indian Medical Gazette*, March, 1902.

ON A PROBABLE WAY BY WHICH THE YOUNG ANKYLOSTOMUM DUODENALE ENTERS THE HUMAN SUBJECT.

By THOS. L. BANCROFT, M.B.Edin., Deception Bay, Queensland.

THE accepted way by which the ankylostoma enter the human body is through the alimentary canal, and cannot be better expressed than in Manson's words: "Should chance so determine, it (*i.e.*, the young ankylostomum) is finally transferred to the human alimentary canal, either in muddy drinking water, or in the mud or dirt adhering to the hands or food dishes of the agriculturist, the brick-maker, or other operative engaged in handling the soil; or, it may be, in earth deliberately eaten by the geophagist."¹ In the *British Medical Journal*, September 14th, 1901, p. 690, there appeared a paper by Professor Sandwith, of Cairo, entitled "Note on the entrance of ankylostoma embryos into the human body by means of the skin."

This paper should be carefully read by all physicians who are called upon to treat cases of ankylostomiasis.

It appears that Dr. Looss, as far back as 1898, stated that the ankylostoma embryos entered the human body by the skin as well as by the alimentary canal, but his statement met with hostile criticism. Recently, however, whilst working in his laboratory with the larvæ of ankylostomum, he accidentally infected himself; the worms entered the skin of his hand and he afterwards suffered from debility and anæmia, and ankylostomum eggs were found in his fæces.

In the Deception Bay district I have had under observation for a number of years two families whose children harbour this parasite; the children have re-infected themselves over and over again in a mysterious manner, but in the light of Dr. Looss's discovery, it would appear that they have done so by means of their feet. They were earth-eaters, but we were never ever able to detect them eating the earth in which the embryos occurred; one child used to eat charcoal, whilst another broke old bricks and picked out certain siliceous pieces, these he pounded, and afterwards swallowed the sand. I am of opinion that persons suffering from ankylostomiasis can bring about a feeling of well-being by swallowing gritty substances; such substances possibly dislodge the parasite from the bowel.

The water they consumed was rain-water, collected from the roofs of the houses and stored in well-constructed tanks; it was pure both at their homes and at the schools they attended.

Neither family used a closet, but each member selected a spot behind bushes, logs, stumps, &c., within a radius of fifty yards from their dwelling; when the spot became foul another one was used, but after rains, when the excreta were disintegrated and

had disappeared from sight, the old locality was again visited, and as all the children went barefooted, it is possible that in treading on the soil containing the young ankylostoma, especially in wet weather, the parasites entered through the delicate skin between the toes.

In addition to the old advice given to sufferers from ankylostoma, viz., "to observe cleanliness, more particularly of the hands, drink only boiled water, and not to eat earth," it would be well to add, "wear boots and use a closet."—*The Australasian Medical Gazette*, February 20th, 1902.

SULPHUR IN THE TREATMENT OF DYSENTERY.

By RAM DHARI SINHA, L.T.M.S.

Medical Officer, Imperial Service Lancers, Jodhpur, Rajputana.

DURING the months of November and December, 1901, I had frequent occasions of treating several cases of dysentery, both in my hospital and in private practice.

I tried sulphur sublimite and Dover's powder, as advocated by Dr. Richmond, of the Imperial Yeomanry, Pretoria, and I can now endorse the gratifying results he claims for his method. The powders were given every four or six hours, either swallowed with a draught of water or mixed with honey or butter.

In two of my cases the powders produced vomiting and nausea, even after the omission of Dover's powder. I sometimes substitute pulv. kurchi., grs. xx., instead of Dover's powder.

Remarks.—The sulphur sublimite, mixed with equal quantity of pulv. holarrhena antidysenterica cortex, has produced the same gratifying results as with pulv. ipecac. co., and sometimes, when idiosyncrasy for ipecac. was prominent, better results.—*Indian Medical Record*, March 5th, 1902.

PLAGUE.

PREVALENCE OF THE DISEASE.

INDIA.—During the two weeks ending March 8th and 15th, the number of deaths from plague reported in India amounted to 23,715 and 25,655 respectively. The majority of this huge total occurred in the Punjab, where during the two weeks in question there died of plague, respectively, 12,544 and 15,090 persons.

In the other districts and cities of India, the number of deaths from plague, during the weeks ending March 8th and 15th, respectively, were: Bombay city, 888 and 856; Bombay Districts, 4,806 and 4,779; Calcutta, 462 and 501; Bengal, 1,235 and 1,289; The North-West Provinces and Oudh, 2,187 and 2,335; Mysore State, 414 and 304.

In the city of Bombay, during the month of March, 1902, the reported cases of plague numbered 4,325, and the deaths from the disease, 3,656. In the Bombay Districts the chief centres of plague were Kaira with 3,875 cases, 2,503 deaths; Broach, 760 cases, 566 deaths; Surat, 592 cases, 449 deaths; Khandesh, 4,345 cases, 3,418 deaths; Nasik, 228 cases,

¹ "Tropical Diseases," p. 540.

185 deaths; Poona, 305 cases, 262 deaths; Satara, 2,927 cases, 2,209 deaths; Sholapur, 755 cases, 619 deaths; Kolaba, 108 cases, 77 deaths; Ratnagiri, 111 cases, 100 deaths; Belgaum, 2,014 cases, 1,536 deaths; Dharwar, 1,483 cases, 1,150 deaths; Hyderabad, 853 cases, 755 deaths; Kathiawar, 497 cases, 321 deaths; Cutch, 304 cases, 233 deaths; Rewa Kantha, 373 cases, 248 deaths; Kolhapur, 1,232 cases, 790 deaths; Janjira, 169 cases, 146 deaths; Aundh, 82 cases, 57 deaths; Savanur, 69 cases, 53 deaths; Baroda, 1,605 cases, 1,144 deaths. Poona city, 78 cases, 78 deaths; Karachi city, 359 cases, 298 deaths. Europeans, 7 cases, 3 deaths.

EGYPT.—The Director-General, Sanitary Department, reports that during the week ending March 30th, 22 fresh cases of plague and 10 deaths from the disease occurred in Egypt. One case was reported from Abousir; 4 cases from Kom-El-Nour; 13 cases and 8 deaths, three of which occurred out of hospital, from Deshneh; 2 cases and 1 death from small villages near Benha; and 2 cases and 1 death from Korachieh. Korachieh is a village in the Santa Markaz of Gharbieh. From enquiries made there it is evident that the infection was carried thither from Tintah, and it appears that the disease broke out first in February among some Bedouins employed in the Domains Estate; instead of at once informing the Sanitary Department of the existence of cases of suspicious disease followed by death among these workpeople, they were dispersed and their huts destroyed, the result being that these wandering Bedouins have disseminated the disease in several small encampments about Benha.

MAURITIUS.—During the week ending March 28th, 5 fresh cases of plague and 4 deaths from the disease occurred in Mauritius. During the week ending April 3rd, neither fresh cases nor any deaths from plague were reported.

CAPE OF GOOD HOPE.—Plague appears to have completely disappeared from Cape Colony.

PLAGUE PRECAUTIONS.

WE notice with a very great deal of pleasure that the Sanitary Authorities are now taking wholesome measures to fight against the plague. The washing of the streets with carbolic disinfectant cannot but prove fruitful of good results and, however expensive the operations may prove, it cannot be too often and too thoroughly carried out. If drastic measures are promptly and properly taken there may be some hope of stamping out this fell disease from the Colony, and should this once be accomplished it might not prove very difficult to keep the disease away. It is a fact that we have disease with us and that it will stay unless every possible facility is given to the very capable and energetic Sanitary Officials who now have to stifle it by their own special methods. We congratulate the Sanitary Authority in having taken the wise steps we have just referred to and would strongly urge that they should entirely yield to the suggestions and recommendations of the gentlemen who have been lately brought to the Colony with the special and sole object of fighting the plague.—*Hong Kong Telegraph*, March 10th, 1902.

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Archives Russes de Pathologie, de Médec. Clinique et de Bacteriologie.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Brooklyn Medical Journal.
Caducée.
Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Esercito
Hong Kong Telegraph.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
Journal of the American Medical Association.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Medical Record.
Medical Review.
Merck's Archives.
New York Medical Journal.
New York Post-Graduate.
Pacific Medical Journal.
Polyclinic.
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Revista de Medicina Tropical.
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The Journal of Tropical Medicine.

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Original Communications.

A THEORY TO EXPLAIN HOW MAN AND THE ANOPHELES ORIGINALLY BECAME INFECTED WITH THE MALARIAL GERM.

By ALBERT J. CHALMERS, M.D., F.R.C.S.
Registrar Ceylon Medical College, Colombo, Ceylon.

INTRODUCTORY.

By kind permission of His Excellency the Right Honourable Sir West Ridgeway, G.C.M.G., K.C.B., K.C.S.I., Governor of Ceylon, I am permitted to offer for publication this attempt to answer the following questions which are often asked by medical and non-medical persons interested in malaria:—

(1) Is it not possible that the infection of man may take place by some other means than by a blood-sucking animal?

(2) How did the cycle of the life history in man and the mosquito start?

It appears to me important to state a theory which will attempt to give reasons why the mosquito is probably the only means of infection at the present time, and also to show how the infection started. The reason why it is important to do this is to try to leave no possible excuse for any person to avoid taking reasonable and simple precautions to prevent himself becoming infected with malarial fever, as the actual demonstration of the fact that the mosquito is the cause has already been given.

These two questions can only be answered by considering the probable evolution of the malarial parasite; and though this of course is only a theory and not open to any proof, yet it will indicate a possible path of infection.

THE EVOLUTION OF THE MALARIAL GERM.

Some of the lowest unicellular forms of animal life, called protozoa, live more or less unprotected in water, though many have evolved various means of defence.

Those unprotected unicellular animals which, in the world's early history, found their way into the digestive canal of higher animals in drinking water would, if they survived the action of the digestive juices (as many do), find themselves able to grow, multiply, and live more easily than those in the outside world; but they would have to produce a large number of spores by the lowest form of reproduction, *i.e.*, the asexual, and these spores would have to be more or less resisting to digestive juices, &c., in order to reproduce the species. In this way a class of animals represented now-a-days by the sporozoa, *i.e.*, animals reproducing asexually by spores, would arise. (They also reproduce sexually at the present time.) But many members of the sporozoa would probably find the action of the digestive juices very irritating, and would therefore be driven to seek further protection, *e.g.*, such as is offered by living in the interior of a cell lining the digestive canal, which is exactly what some members of the Coccidiidæ, *viz.*, *Coccidium Oviforme* in man and rabbits, do at the present day. This animal is known to reproduce itself asexually and sexually very much in the same way as the malarial germ does, except that it all takes place in the alimentary canal of its host, and that infection is by means of substances swallowed.

Other members of the Coccidiidæ, however, passed either through the cells, or more likely between the cells lining the digestive tract, into mucous membrane of the digestive tract which lies under these cells. (At the present day this is actually done by some Coccidiidæ.) There they would find new enemies waiting for them, *viz.*, the so-called white blood cells or phagocytes, and to further protect themselves they wandered (*e.g.*, *Coccidium Oviforme*), some into the liver, while others entered the capillary blood-vessels and lay free in the blood stream, just as the spores and the young malarial germs do to-day; but even here they would be liable to be attacked by the phagocyte, and to avoid them still further they entered the red blood cell, where they found a haven

of rest till the discovery of quinine. Those which did not find this haven would eventually die out in the struggle for existence. The unicellular animals who did this are to-day called *Hæmamœbidæ*, and are known to cause disease in man, cattle, several birds (pigeons, larks, sparrows, starlings, owls, screech owls), and in frogs.

Once in the red blood cells they will circulate all over the body, and at some time or other will be found in the skin.

THE INFECTION OF THE MOSQUITO.

By the time that these *Hæmamœbidæ* had undergone their evolution in the tissues of warm-blooded animals the gnats would have evolved, and would have acquired their peculiar mouth apparatus for piercing tough structures and for sucking juices; and the females of certain of these gnats would have acquired the habit of taking in warm blood for the development of their eggs.

Consequently sooner or later the malarial parasites would find themselves transferred from the tissues of the skin of man to the stomach of a mosquito. In this new habitat they would be liberated from the red blood cell (*e.g.*, the present day quartan and tertian gametocytes), probably by their own action. On finding themselves unprotected in this new and probably not too congenial abode, they would hasten to do what all unicellular animals try to do under such circumstances, *viz.*, "conjugate." The development of such motile threads as the male spermatozoa would accelerate this act, as they would be chemically attracted by the female cells, and this might be highly beneficial if the juices in the stomach of the mosquito were unfavourable, as, for example, they appear to be in *Culex*, and many other blood suckers.

The energy introduced by the male element into the female would induce the result of the union (the vermicule) to wander about to seek some protection from these irritating juices.

Consequently those vermicules would survive which passed between the cells of the stomach of the mosquito, even as their ancestors had done in the stomach and intestines of higher animals, but finding nothing much on the other side of these cells (instead of a nutritious mucous membrane), they would, as other protozoa do in troubled times, become encysted (zygote), and break up into spores (blastophores and blasts), and these, by bursting of the capsule, would be liberated into the cavity surrounding the stomach of the mosquito, and would go from thence into its various organs, including the salivary glands, and from these to its saliva, and hence to man again.

The *hæmamœba* of malaria in man develops in a mosquito called the *Anopheles*, which is not very common, that of the bird in *Culex pipiens*, which is common, and that of Texas fever in cattle in a tick. When once such a suitable course of life history is entered upon, those germs which fail to carry it out die, *i.e.*, the germs of malarial fever in such *Culex* die; consequently only one species is left (the most suitable for the life of the parasite), by means of which it can be propagated, *viz.*, the *anopheles* mosquito. In this way an explanation of the original mode of entry of the malarial germ into the human

being and of the original method of infection of the mosquito is possible. The vertebrate host, including man, must have been the original host, because in him the adult stage of the animal's life history is found; the mosquito is of importance in the sexual production and in propagating the parasite.

Summary.—By the action of the digestive juices the protozoa which enter the digestive tract of higher animals find themselves weeded out into those which can resist the juice with immunity and those which cannot. Those of the latter which do not seek further protection die out, the others seek protection by getting under cover of the lining cells of the digestive tract. Those which pass under the cells into the mucous membrane find new foes, to avoid which they enter the red blood cells. The gnat now swallows the red blood cell containing the parasite. The parasite again finds itself in a digestive tract with juices, and again protects itself by passing under the mucous membrane, and to reproduce its species breaks up into a large number of blasts.

SMALL-POX AND VACCINATION IN BANGKOK.

By P. A. NIGHTINGALE, M.D. (Harrogate).

Formerly of Bangkok, Siam.

THE Asiatics resident in the capital of Siam (whose estimated population is over 400,000) form a striking object lesson to the "conscientious objector" in Britain.

Living for the most part in a state of *jus naturale*, in the original sense of the term, they are accustomed to the dreadful ravages of most of the more deadly tropical diseases and accept them as part of their fate without a murmur, knowing that under the existing local conditions there is not much to be done against cholera, dysentery and malarial fever.

But when it comes to small-pox they are on different ground, and fully realise the marvellous prophylactic value of "efficient" vaccination. Though there is no law dealing with the subject the upper classes almost invariably employ European medical men to vaccinate their children during the cooler months of the year, while the middle class save up their money and buy the lymph themselves—the mother or Buddhist priest performing the operation. For the lower and coolie class the Government is beginning to open free vaccination stations, both in Bangkok and the provinces, which will be taken more advantage of as their existence gets more widely known.

A Siamese mother will insist in a way which ought to put to shame her more enlightened sisters over here, that four large "marks" be made—on the right arm for girls, and on the left for boys—and will tell you that if they all "take" there is no need for re-vaccination later on, but if only one or two of the insertions are successful they will certainly wish for the operation to be repeated within a very few years.

As their garments are scanty in the highest degree, there is seldom any trouble from the introduction of foreign material into the pustules from foul coverings;

about the fourth day they will neatly cover up each mark with some native paste largely composed of turmeric, which seems to allay all irritation, and is only finally removed when the arm is quite healed.

Once vaccinated, they lose all fear of the disease, and will cheerfully live, and even sleep, with an affected case in any stage.

Of course small-pox is always endemic, and from time to time epidemic, in Bangkok, but (in the absence of all statistics) judging from the yearly number of cases which come under one's personal supervision, it is undoubtedly gradually dying out, though the general sanitation connected with the lives of the lower classes is not improving at the same rate.

Dr. Campbell Highet, the Medical Officer of Health, hopes soon to establish an animal vaccine station, and when this is affected many of the present difficulties connected with the supply of lymph will be done away with, and voluntary vaccination undoubtedly largely increase.

Altogether a striking object lesson is taking place in Bangkok, a lesson which it is only a pity that "conscientious objectors" in Britain cannot examine for themselves, instead of being fed up on literature written by persons who have never practically studied small-pox in its far Eastern home, among a people who are bound by no law save a natural desire to live, and who are gradually "working out their own salvation."

A PECULIAR AND UNDESCRIBED AFFECTION OF THE NOSE.

By Dr. J. C. MITCHELL.
Grahamstown, Cape Colony.

AN interesting observation, illustrated by the photograph I am sending, is a form (apparently undescribed) of disease affecting the nose in natives. I was fortunate to see in the lock wards of the hospital three cases which were diagnosed as syphilis. In the girl on the left in the photo the diagnosis is probably correct. The condition healed under the exhibition of K.I., and the scar is typically syphilitic.

The right hand case may be taken to show an early condition of the central one. It had lasted some months, and according to the patient it had commenced by the formation of small, hard nodules under the skin, which gradually enlarged, till at the time of examination they were of the size of small peas. A serous fluid exuded from the surface of the older ones which formed a crust over the surface. There was no ulceration. In the central case the condition has gone on to ulceration and much loss of tissue. The nodular condition still persists on the skin, covering the ala nasi and tip, but the septum and upper lip are much eroded. The floor of the ulcer, light coloured in the picture, presented numerous raw protuberant granulations quite unlike a syphilitic ulcer.

The condition in the early and later stages is not painful and the nodules are almost anæsthetic. In neither of these cases were the mucous membranes of the mouth, fauces, or pharynx affected, though that of the anterior part of the nose was. There was further no history or sign of syphilis in either of these

cases. I made arrangements to have pieces excised for excavation, but both girls left before this could be done. One—the central one—has returned with the condition unchanged, so that I may have an opportunity of observing her further.

The appearance and history, together with the marked resistance to iodide of potassium, is against syphilis. Is it rhinosclerosis?

PLAGUE PROPHYLAXIS IN FORMOSA.

By JAMES L. MAXWELL, M.D.Lond.

Now that the bubonic plague bids fair to become pandemic in its grasp, and the danger seems no longer to be confined to Eastern lands, any new method of treatment or new application of old methods may well be seriously considered. This alone can be my excuse for touching on a subject about which so much has lately been penned. In brief, I wish to describe the prophylactic measures adopted by the Japanese medical authorities in the Island of Formosa. My remarks will principally apply to the old capital of the Island, Tainan, in which I am myself resident, and in which I have watched the measures here described in their process of action.

Your readers will remember that the Japanese have only possessed the island since the termination of the late Chino-Japanese war, and even then it had all to be taken at the point of the sword. As a result of this, the conquerors have been able to deal with the natives without any regard to their natural prejudices, which I need hardly point out could scarcely be done in a civilised state with ancient laws. I feel at once this is the weakness of the system I am about to describe, and probably would prove fatal to it in most other places, though I see no reason why it should not be enforced in single places like Hong-Kong, or among part of the population, as say, among the natives in Cape Town.

The system consists in the compulsory inoculation of plague toxins, the preparation used being that of Professor Kitasato, prepared in his laboratories in Tokyo. As this differs from Professor Haffkine's preparation in some very important particulars, it would be well at once to mention its method of manufacture. The bacillus taken directly from an infecting bubo, or from the blood of a septicæmic case, is cultivated on sloped agar tubes in an incubator, at the temperature of 31 to 32 C., for from forty to seventy hours.

The surface of the cultures is then scraped, the scrapings pounded in a sterilised mortar, and sterilised normal salt solution is added to a fixed dilution. The solution is then filtered through a fine wire sieve, and the filtrate heated to 60 per cent. for thirty minutes. Carbolic acid is added till it is present in the solution to the strength of 5 per cent. The preparation is kept in ice, and cultures and animal inoculations are made to prove its sterility. It is then sealed in sterilised bottles and is ready for use.

In addition to these differences in preparation from Haffkine's fluid, there are also very marked differences in its physical action. I have not personally

experienced or observed the use of Haffkine's fluid, but I have been told by medical men who have experienced and observed its use that its action is nearly always very distressing to the patients, sometimes really causing severe and prolonged physical effects. I have myself been thrice inoculated, and have seen numerous cases of inoculation with Kitasato's fluid, and have never seen a patient confined to bed with it, or seriously inconvenienced in any way.

Tainan, to which city I shall principally refer, is a Chinese city of nearly 50,000 souls. In saying it is Chinese I mean to imply that it has the narrow streets, the low filthy shops, and all the other objectionable features of the cities of the Celestial Empire. True, it never was as bad as its neighbours on the mainland, and the Japanese have made many improvements both in the way of cleanliness and in driving a few large roads through the city. But it still remains a place where the best disinfecting measures would be utterly thrown away—an Augean stable which it would take a Hercules indeed to cleanse. Under these circumstances the authorities have very wisely abstained from pushing disinfecting measures to any great extent, contenting themselves with a few general regulations and these inoculations, which I will now describe.

At first an attempt was made to isolate the relations of the patients by sealing the houses in which a case had occurred; but this was never a very successful measure, and after a time was more or less dropped.

After a few sporadic cases during January and February, 1901, the real epidemic began about the earlier part of March and raged till the middle of July. At the height of the disease there was a known death-rate of nearly fifty a day, and as many deaths were concealed, bodies being buried in the houses, gardens, and, indeed, every conceivable place, to escape the eye of the officials, this death-rate should no doubt have been put at a decidedly higher figure.

The compulsory inoculations were begun rather late in the season or a still larger proportion of the population would have been inoculated. As it was, however, more than 15,000 persons were inoculated in the city alone. The numbers are therefore quite large enough to avoid the errors which smaller figures might lead one into. Some may suggest that the type of disease was mild, but I would negative this, the mortality rate I have already mentioned, and that I shall hereafter mention, are both high, and the cases I saw were, many of them, very severe. The first case to which I was called was a child who became ill shortly before mid-day, and died at 9 p.m. the same night, and I saw many other fulminating cases.

The inoculations were carried out by Dr. Tsukiyama, the senior Government physician in Tainan, and by his assistant, Mr. Kamachi. To the latter I am much indebted for the figures and statistics in this article.

For the purpose of inoculation the city was divided into a number of squares, one of these squares being dealt with at a time, the desire of the authorities being to inoculate as far as possible every one in the city three times, at intervals of about a week. The people in the locality being dealt with were brought into a conveniently situated temple, or other large building, and there inoculated, the men and women

being separately treated. This house to house method had the advantage that it allowed the authorities to discover concealed cases, which, as I have already said, were far from being uncommon. As by this house to house visitation the men were particularly liable to escape inoculation, a second method was adopted of arresting passers-by or making surprise visits to the markets. Very careful records of all the inoculated were kept, and should the same person be again arrested his previous records were referred to, and the fact of his second or third inoculation noted. By these methods over 15,000 people in the city alone were inoculated, for the most part, on three successive occasions.

As far as the inoculations themselves go, they are simplicity itself. A spot is selected in the muscles of the back, between the vertebral borders of the two scapulae, cleansed with an antiseptic wash and with ether, and the inoculation then made deeply by means of an ordinary hypodermic syringe. The quantity injected is 1 gramme on the first occasion, 1.5 grammes on the second, and 2 grammes on the third, the proper interval between inoculations being one week. I never saw or heard of any septic trouble in connection with the sites of injections, but once or twice saw a few urticarial spots about the situation of the puncture. As I was attending a good many plague cases myself I gladly submitted to the process, so in mentioning it I am speaking from personal experience. The immediate pain on injection is quite infinitesimal. On each occasion I was inoculated about mid-day. During that afternoon and evening I had a slight aching and stiffness in the muscles involved, accompanied by some swelling at the site of the injection; by the next day this was passing off, but I suffered from slight malaise, not sufficient to prevent my doing my ordinary work. By the following day I was perfectly well again: this was repeated after each inoculation. Personally I suffered from no pyrexia, though I understand that a temperature of 100° F. is not rare. I never saw anyone confined to bed as a result of the inoculations.

Before any of the statistics had been published, I had formed very decidedly favourable opinions with regard to the value of the inoculations from my own observation of my patients. I attended many cases of plague, most of whom met with the usual termination of this fatal disease; yet, though one-third of the city had been injected I only came across two cases of the disease in inoculated persons. In one of these, a young man, the disease was so mild that the patient was hardly confined to bed. The other case was that of a young woman, and was much more severe, but terminated in convalescence, delayed by an indolent sinus following a groin bubo which is only just healed. She, however, had only been inoculated once.

I append to this paper statistical tables dealing with the city of Tainan and then with a few of the neighbouring villages, where also inoculations were carried out, the third table being a summary of the preceding two. I would only like to call your special attention to two of the figures in the last table, the percentages of deaths and the percentages of attacks. A moment's calculation will show that had the attacks among

those inoculated been in the same proportion as among those who were not inoculated, there would have been 578 cases in the place of 41; while had the inoculated died in the same proportion as the non-inoculated, there would have been 34 deaths in the place of 22. These figures speak for themselves and follow, too, a common rule in this class of preventative inoculations, viz., that their effect is principally prophylactic and to a very much smaller extent curative. Statistics, as we all know, are not to be relied on to too great an extent, especially where very small numbers are concerned, but here, where our totals work out into many thousands, the suggestion that we have too few by which to judge can hardly be regarded as tenable. Of course, one must allow that no doubt many attacks and many deaths in the city were concealed, and so escaped these tables, but there can be no reason to suppose that these deaths would militate against the statistics here brought forward. Rather it would be to the advantage of the people to conceal deaths among those who had managed illegally to avoid inoculation, and to make the most of those who had been inoculated and yet died, as it need hardly be said the prejudiced Chinese view with great distaste the compulsory inoculation.

TAINAN CITY.

| Total Population | Number Inoculated | Number not Inoculated | Attacked by Plague | | Number of Deaths | | Percentage of Deaths among those Attacked | | Percentage of those afterwards Attacked by Plague | |
|------------------|-------------------|-----------------------|--------------------|----------------|------------------|----------------|---|----------------|---|----------------|
| | | | Inoculated | Not Inoculated | Inoculated | Not Inoculated | Inoculated | Not Inoculated | Inoculated | Not Inoculated |
| 47,382 | 15,678 | 31,704 | 30 | 1,130 | 13 | 948 | 43.33 | 83.89 | .19 | 3.64 |

NEIGHBOURING VILLAGES.

| | Total Population | Number Inoculated | Number not Inoculated | Attacked by Plague | | Number of Deaths | | Percentage of those afterwards Attacked by Plague | |
|-----------|------------------|-------------------|-----------------------|--------------------|----------------|------------------|----------------|---|----------------|
| | | | | Inoculated | Not Inoculated | Inoculated | Not Inoculated | Inoculated | Not Inoculated |
| Anping .. | 5,378 | 605 | 4,773 | 1 | 21 | 1 | 17 | .16 | .44 |
| Tai-ko .. | 631 | 124 | 507 | 2 | 87 | 2 | 69 | 1.61 | 17.16 |
| Tsan-bun | 158 | 72 | 86 | 1 | 8 | 1 | 5 | 1.39 | 9.30 |
| Kagi .. | 19,039 | 2,075 | 16,964 | 2 | 231 | 2 | 199 | 0.9 | 1.36 |
| Poa-ka .. | 6,677 | 2,097 | 4,580 | 5 | 170 | 3 | 148 | 2.4 | 3.71 |

TOTAL STATISTICS FOR SOUTH FORMOSA.

| Total Population | Number Inoculated | Number not Inoculated | Attacked by Plague | | Number of Deaths | | Percentage of Deaths among those Attacked | | Percentage of those afterwards Attacked by Plague | |
|------------------|-------------------|-----------------------|--------------------|----------------|------------------|----------------|---|----------------|---|----------------|
| | | | Inoculated | Not Inoculated | Inoculated | Not Inoculated | Inoculated | Not Inoculated | Inoculated | Not Inoculated |
| 79,265 | 20,651 | 58,614 | 41 | 1,647 | 22 | 1,386 | 56.09 | 84.15 | .19 | 2.80 |

To sum up. By a method of inoculation easily applied and causing a minimum of discomfort a very large reduction has been made in the numbers attacked by plague in this place, and a decided reduction in the number of the deaths among those attacked.

As I have already said, the exceptional political circumstances of this island make it possible to carry out compulsory inoculation in a way which probably could not be done in many places. In isolated colonies, however, this might be possible, at least among the native populations, and a wide circle of "contacts" might easily be treated when sporadic cases break out in our own cities.

THE DURATION OF THE LATENCY OF MALARIA AFTER PRIMARY INFECTION, AS PROVED BY TERTIAN OR QUARTAN PERIODICITY, OR DEMONSTRATION OF THE PARASITE IN THE BLOOD.

By DR. ATTILIO CACCINI,

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(Translated from the Italian by St. Clair Thomson, M.D.Lond., F.R.C.S.)

(Continued from page 122.)

PART II.

ON RELAPSES AT SHORT INTERVALS.

THE factors of which we spoke in the preceding chapter, manifest, as we said, their influence particularly in determining the relapses at long intervals.

On the other hand, on the relapses at short intervals the effect of treatment by quinine is very much more important, and hence I avail myself of this fact to divide into several groups and sub-groups the results obtained in regard to relapses at short intervals, and I will first refer to spring tertian, then to the quartan, and then to the malignant tertian.

I will commence by saying that the quinine treatment influences the quota of relapses.

In regard to the treatment, malarial patients can be divided into two great categories:—

- (A) Patients who have had quininism.
- (B) Patients who have never had quininism.

Chapter 1.—Spring Tertian.

(1) ON THE RELAPSES AT SHORT INTERVALS IN PATIENTS WHO HAD QUININE IN THE FIRST INFECTION.

Commencing with what takes place in individuals submitted to the quinine treatment, we must make a clear distinction between those who have been submitted to rational administration of quinine, and those who have carried out the treatment in a more or less irregular manner.

Hence I divide my cases into three categories:—

- (A) Cases in which the quinine treatment was carried out early and systematically.¹

¹ By systematic treatment, I mean the administration of quinine before the febrile access, in such a manner that it is in circulation exactly at the moment of sporulation of the parasites.

(B) Cases with a retarded systematic treatment—that is, cases in which the treatment was systematic, although initiated late and after the outbreak of many attacks.

(C) Cases treated without system—that is, in which the administration of the salt of quinine was carried out every day, but without any precision, and in variable doses; either administered quite irregularly, or during the febrile acme, or during the period of perspiration.

From a tabular exposition of these cases it is at once seen how with the intensity and quality of the quinine treatment the malarial infection varies in its behaviour, both in respect to the appearance of the relapses, and in the case of relapses, in respect to their gravity, precocity, and number.

GENERAL EXPLANATION OF THE MANNER IN WHICH THE TABLES WERE COMPILED.

(A) In the first vertical column are indicated the number of days during which the several patients were under observation.

(B) In the second vertical column I indicate the number of patients studied in correspondence to the length of observation.

(C) In the successive vertical lines I indicate the number of patients who relapsed, and particularly in the first column, those who relapsed after five days of observation: in the second column those who relapsed after six days, and so on after seven, eight, &c., days, as is indicated by the numbers in the horizontal line at the head of the Table.

Thus, for example, in Table No. 3, we were able to follow for seven days, 21 patients, of whom 2 relapsed on the fifth day of apyrexia, the others disappeared from observation without having presented a relapse, and proceeding with eight days of observation 20 cases were followed; of which 1 relapsed upon the sixth day, and 1 on the eighth day of apyrexia, and so on for every Table.

TABLE I.

GENERAL CONSIDERATIONS—Year 1900.

Malignant Tertian.

| | June | July | Aug. | Sept. | Oct. | Nov. | Dec. | Total |
|--------------|------|------|------|-------|------|------|-------|-------|
| Primary .. | 238 | 424 | 510 | 176 | 130 | 1 | 1,379 | |
| Relapsing .. | 10 | 317 | 182 | 217 | 120 | 100 | 946 | |
| Total .. | 248 | 741 | 692 | 393 | 250 | 101 | 2,325 | |

Spring Tertian.

| | June | July | Aug. | Sept. | Oct. | Nov. | Dec. | Total |
|--------------|------|------|------|-------|------|------|------|-------|
| Primary .. | 22 | 110 | 55 | 78 | 153 | 10 | 3 | 431 |
| Relapsing .. | 35 | 14 | 37 | 14 | 88 | 100 | 75 | 363 |
| Total .. | 57 | 124 | 92 | 92 | 241 | 110 | 78 | 794 |

Quartan.

| | June | July | Aug. | Sept. | Oct. | Nov. | Dec. | Total |
|--------------|------|------|------|-------|------|------|------|-------|
| Primary .. | 8 | 5 | 71 | 120 | 50 | 6 | 260 | |
| Relapsing .. | 5 | 15 | 1 | 11 | 23 | 40 | 60 | 155 |
| Total .. | 5 | 23 | 6 | 82 | 143 | 90 | 66 | 415 |

| | June | July | Aug. | Sept. | Oct. | Nov. | Dec. | Total |
|----------|------|------|------|-------|------|------|------|-------|
| Complete | 62 | 395 | 839 | 866 | 777 | 450 | 245 | 8,534 |

TABLE II.

CASES OF QUARTAN WHICH RELAPSED AFTER THE INTERVENTION OF ONE OF THE ORDINARY DETERMINING FACTORS.

| General observations | Attacks before the finishing of the fever | Duration of the apyrexial period before the intervention of the determining factor | Duration of the apyrexial period after that factor | Determining factor | Febrile attacks during the relapse |
|-----------------------------|---|--|--|-------------------------------------|------------------------------------|
| 1 Peasant, aged 28, primary | 6 | 30 days | 2 days | Contusion on the thorax and abdomen | 3 |
| 2 Peasant, aged 40. | 5 | 25 days | 2 days | Rain | 5 |
| 3 Peasant, aged 27. | 9 | 26 days | 2 days | Rain | 2 |
| 4 Student, aged 16. | 7 | 24 days | 2 days | Cold bath .. | 3 |
| 5 Peasant, aged 19. | 5 | 19 days | 2 days | Cold bath .. | 2 |
| 6 Peasant, aged 48. | 10 | 22 days | 36 hours | Excessive heat | 2 |
| 7 Peasant, aged 21. | 6 | 27 days | 29 hours | Drunkenness, fighting and wounds | 2 |
| 8 Peasant, aged 26. | 6 | 18 days | 2 days | Chloroform .. | 3 |

EXPLANATION OF TABLE II.

None of these patients had ever taken quinine.

TABLE IIA.

GENERAL CONSIDERATIONS—Year 1901.

Malignant Tertian.

| | June | July | Aug. | Sept. | Oct. | Nov. | Total |
|--------------|------|------|------|-------|------|------|-------|
| Primary .. | 7 | 67 | 429 | 225 | 136 | 82 | 896 |
| Relapsing .. | 11 | 192 | 159 | 96 | 25 | 483 | |
| Total .. | 7 | 78 | 621 | 384 | 232 | 57 | 1,379 |

Spring Tertian.

| | June | July | Aug. | Sept. | Oct. | Nov. | Total |
|--------------|------|------|------|-------|------|------|-------|
| Primary .. | 11 | 160 | 293 | 161 | 53 | 7 | 685 |
| Relapsing .. | 66 | 89 | 249 | 126 | 26 | 15 | 571 |
| Total .. | 77 | 249 | 542 | 287 | 79 | 22 | 1,256 |

Quartan.

| | June | July | Aug. | Sept. | Oct. | Nov. | Total |
|--------------|------|------|------|-------|------|------|-------|
| Primary .. | 3 | 110 | 128 | 22 | 263 | | |
| Relapsing .. | 14 | 22 | 20 | 44 | 72 | 5 | 177 |
| Total .. | 14 | 25 | 20 | 154 | 200 | 27 | 440 |

| | June | July | Aug. | Sept. | Oct. | Nov. | Total |
|-------------------|------|------|-------|-------|------|------|-------|
| Complete Total .. | 98 | 352 | 1,183 | 825 | 511 | 106 | 3,075 |

TABLE III.

SPRING TERTIAN TREATED SYSTEMATICALLY AND EARLY.

| Days of Observation | No. of Cases | Duration of the Latent Period Calculated in Days | | | | | | | | | | | | | | | | |
|---------------------|--------------|--|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|
| | | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | | | |
| 7 | 21 | 2 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 8 | 20 | 2 | 1 | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 10 | 10 | .. | 1 | 1 | 1 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 15 | 10 | .. | 1 | 1 | 1 | 1 | 1 | .. | 2 | 1 | 1 | .. | .. | .. | .. | .. | .. | .. |
| 21 | 6 | .. | 1 | 1 | .. | 1 | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 25 | 9 | .. | .. | 1 | .. | .. | 1 | .. | 1 | 1 | .. | .. | .. | .. | 1 | .. | .. | .. |
| 30 | 10 | .. | .. | 1 | .. | .. | .. | 1 | .. | .. | .. | 1 | 1 | 1 | 1 | .. | .. | .. |
| 35 | 10 | .. | .. | .. | .. | .. | 1 | .. | .. | .. | 1 | 1 | 1 | 1 | 1 | .. | .. | .. |
| 40 | 7 | .. | .. | .. | .. | .. | .. | .. | 1 | .. | 1 | .. | .. | .. | 1 | 1 | .. | .. |
| 45 | 6 | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | 1 | .. | 1 | 1 | 1 | .. | .. |
| 50 | 9 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 55 | 6 | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | 1 | .. | .. | .. | .. | .. |
| 60 | 5 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | 1 | .. | .. | .. |
| 90 | 3 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 100 | 3 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 110 | 2 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 120 | 2 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 130 | 4 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 |
| 140 | 2 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Total per day .. | | 4 | 4 | 5 | 3 | 3 | 3 | 2 | 4 | 4 | 3 | 4 | 3 | 4 | 10 | | | |

TABLE IV.

SPRING TERTIAN WITH DELAYED SYSTEMATIC TREATMENT.

| Days of Observation | No. of Cases | Duration of the Latent Period Calculated in Days | | | | | | | | | | | | | | | | |
|---------------------|--------------|--|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|
| | | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | | | |
| 16 | 30 | 2 | .. | .. | 2 | .. | .. | 1 | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. |
| 17 | 22 | .. | 2 | .. | .. | 2 | .. | 2 | .. | .. | .. | 1 | 1 | .. | .. | .. | .. | .. |
| 18 | 20 | .. | .. | 1 | .. | 1 | .. | .. | .. | 2 | 2 | .. | .. | 2 | .. | .. | .. | .. |
| 19 | 26 | .. | .. | .. | .. | .. | 2 | .. | 2 | .. | .. | .. | .. | .. | 2 | .. | .. | .. |
| 20 | 16 | .. | .. | .. | .. | .. | .. | .. | .. | 2 | .. | .. | 2 | .. | .. | .. | .. | .. |
| 23 | 20 | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | 1 | .. | 4 | 3 | .. | .. | .. |
| 24 | 24 | .. | .. | .. | .. | .. | .. | .. | 1 | .. | 1 | 1 | .. | 1 | .. | .. | .. | .. |
| 25 | 12 | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | 1 | .. | 1 | .. | .. | .. | .. |
| 26 | 16 | .. | .. | .. | .. | .. | .. | .. | 1 | .. | 1 | 1 | .. | 1 | .. | .. | .. | .. |
| 27 | 15 | .. | .. | .. | .. | .. | .. | .. | 1 | 1 | .. | .. | .. | 1 | .. | .. | .. | .. |
| 29 | 10 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | 2 | 1 | .. | .. | .. | .. |
| 32 | 10 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 2 | 2 | 1 | .. | .. | .. |
| 33 | 9 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | 1 | 1 | .. | .. | .. | .. |
| 34 | 10 | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | 1 | .. | 1 | .. | .. | .. | .. |
| 36 | 7 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | 1 | 1 | .. | .. | .. | .. |
| 40 | 12 | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | 1 | .. | 1 | .. | .. | .. | .. |
| 45 | 10 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 2 | 1 | .. | .. | .. | .. |
| 50 | 9 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | 1 | .. | .. | .. | .. |
| 55 | 4 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. |
| 60 | 3 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. |
| 65 | 6 | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. |
| 90 | 2 | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. |
| 100 | 2 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 110 | 2 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 120 | 2 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 130 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 140 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. |
| Total per day .. | | 2 | 2 | 1 | 2 | 3 | 2 | 3 | 4 | 5 | 6 | 8 | 13 | 14 | 28 | | | |

EXPLANATION OF TABLE III.

None of those patients had previously taken quinine before entering the hospital. They were given quinine on alternate days in doses of 1½ to 2 grammes per day, in three or four doses, about half-an-hour's interval between them, starting the administration of the remedy three hours before the commencement of the fever.

In those who remained more than ten days permanently in the hospital the treatment was suspended after seven days. This intense method of treatment never produced any disturbance except occasionally some vomiting. To obviate this drawback we administered the remedy in the same dose by endo-muscular injections, always before the initiation of the fever. The cure was recommenced every time that a relapse set in.

EXPLANATION OF TABLE IV.

None of these patients had ever taken quinine before entering the hospital. The quinine was administered on alternate days in the same method as that for the patients described in the preceding table. In rare cases we were forced to have recourse to injections of quinine, but as a rule the remedy was administered by the mouth. The treatment was renewed on the outbreak of relapse.

TABLE V.

SPRING TERTIAN WITH DAILY QUININE.

| Days of Observation | No. of Cases | Duration of the Latent Period Calculated in Days | | | | | | | | | | | | | | | | |
|---------------------|--------------|--|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|
| | | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | | | |
| 6 | 110 | 10 | 10 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 8 | 112 | 10 | 9 | 10 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 10 | 140 | .. | 8 | 10 | 15 | 9 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 12 | 150 | 1 | 6 | 10 | 10 | 11 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 14 | 100 | 3 | 10 | 10 | 9 | 12 | 11 | 9 | 15 | 10 | .. | .. | .. | .. | .. | .. | .. | .. |
| 15 | 90 | 10 | 6 | 4 | 9 | 12 | 2 | .. | 9 | 6 | 4 | .. | .. | .. | .. | .. | .. | .. |
| 16 | 91 | 10 | 9 | 6 | 10 | 11 | 10 | 14 | 10 | 7 | 3 | 1 | 2 | .. | .. | .. | .. | .. |
| 18 | 42 | 99 | 1 | 7 | 6 | 8 | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. |
| 20 | 10 | 1 | 2 | 1 | 1 | 1 | 1 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 21 | 18 | 1 | 1 | 3 | 2 | 1 | 1 | 1 | 2 | 1 | .. | 1 | .. | 1 | .. | .. | .. | .. |
| 22 | 16 | 1 | 1 | 2 | 1 | 1 | 2 | 3 | 1 | 1 | 1 | .. | .. | 1 | .. | .. | .. | .. |
| 24 | 10 | 2 | 2 | 1 | 1 | 1 | 1 | 1 | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. |
| 26 | 12 | 2 | 1 | 1 | 2 | 1 | 2 | 3 | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. |
| 28 | 15 | 2 | 3 | 2 | 1 | .. | 1 | 1 | .. | 1 | .. | 1 | .. | 1 | .. | .. | .. | .. |
| 30 | 10 | 1 | 2 | 1 | .. | 1 | .. | 1 | 1 | .. | .. | 1 | .. | 1 | .. | 1 | 1 | 1 |
| 31 | 19 | 1 | 2 | 2 | 1 | 1 | 1 | .. | 1 | .. | 1 | .. | 1 | .. | .. | 1 | .. | .. |
| 34 | 10 | 1 | 1 | 2 | 1 | .. | 1 | .. | 1 | .. | .. | .. | 1 | .. | .. | 1 | .. | .. |
| 36 | 10 | .. | 1 | 2 | 1 | .. | 2 | 1 | 1 | 1 | .. | .. | .. | .. | .. | .. | .. | .. |
| 41 | 10 | 2 | 1 | 1 | 1 | 1 | 1 | .. | 1 | .. | 1 | 1 | 1 | 1 | 1 | .. | .. | .. |
| 50 | 10 | 1 | 1 | 1 | .. | 1 | 1 | .. | 1 | 1 | .. | 1 | 1 | 1 | .. | .. | .. | .. |
| 60 | 9 | .. | 1 | 1 | 1 | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 70 | 8 | 1 | .. | .. | 1 | .. | 1 | .. | 1 | .. | 1 | .. | .. | .. | .. | .. | .. | .. |
| Total per day | | 159 | 78 | 77 | 73 | 72 | 37 | 37 | 42 | 30 | 13 | 5 | 5 | 3 | 2 | | | |

EXPLANATION OF TABLE V.

These patients had never taken quinine. The remedy was administered in doses varying from 1 to 2 grammes per day by the mouth, without selecting any special period. The treatment, as a rule, was suspended after seven to ten days, and it was renewed on the outbreak of a relapse.

EXPLANATION OF TABLE VI.

These patients had never taken quinine before entering the hospital. It was administered in doses of 1½ grammes during the febrile acme, then repeated on successive days at the same hour. In this way the quinine was repeated every seven days, and resumed in the same method on the outbreak of a relapse. Of thirty-eight patients who relapsed, in ten of them we were able to follow more than one relapse. These relapses take place always within from five to ten days without a fixed period.

TABLE VI.

CASES OF SPRING TERTIAN WITH QUININE DURING THE FEBRILE ACME.

| Days of Ob- serva- tion | No. of Cases | Duration of the Latent Period Calculated in Days | | | | | | | | | | | | | | | | |
|-------------------------------|-----------------|--|----|----|----|----|----|----|----|----|----|----|----|----|----|----|--|--|
| | | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | | | |
| 7 | 6 | 1 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | |
| 8 | 9 | 1 | 2 | 2 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | |
| 9 | 7 | 1 | 1 | 2 | 2 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | |
| 11 | 4 | 1 | 1 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | |
| 12 | 2 | 1 | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | |
| 13 | 5 | 1 | 1 | .. | 1 | 1 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | | |
| 15 | 2 | 1 | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | |
| 17 | 3 | 1 | .. | 1 | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | | |
| 18 | 3 | 1 | .. | 1 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | |
| 19 | 3 | 1 | 1 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | |
| 20 | 1 | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | |
| 21 | 1 | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | |
| 23 | 1 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | |
| 24 | 1 | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | |
| 25 | 1 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | |
| 26 | 1 | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | |
| Total per day .. | | 12 | 10 | 9 | 5 | 2 | 2 | .. | .. | .. | .. | .. | .. | .. | .. | .. | | |

TABLE VII.

SPRING TERTIAN; QUININE DURING SWEATING STAGE.

| Days of Ob- servaion | No. of Cases | Duration of the Latent Period Calculated in Days | | | | | | | | | | | | | | | | | |
|-------------------------|-----------------|--|----|----|----|----|----|----|----|----|----|----|----|----|----|----|--|--|--|
| | | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | | | | |
| 7 | 7 | 1 | 2 | 3 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | | |
| 8 | 8 | .. | 2 | 2 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | | |
| 9 | 5 | 2 | 1 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | | |
| 10 | 6 | 1 | 2 | 1 | 1 | 2 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | | |
| 12 | 4 | 1 | 2 | 2 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | | |
| 14 | 3 | 2 | 2 | 1 | 1 | 1 | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | | | |
| 17 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | | |
| 20 | 1 | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | | |
| 21 | 2 | 1 | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | | |
| 23 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | | |
| 26 | 1 | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | | |
| 28 | 2 | .. | .. | .. | .. | .. | .. | 1 | .. | 1 | .. | .. | .. | .. | .. | .. | | | |
| 11 | 2 | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | | |
| 31 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | | |
| 32 | 3 | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | | | |
| 36 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | | |
| 90 | 2 | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | | | |
| 91 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. | | | |
| Total per day .. | | 8 | 13 | 11 | 3 | 3 | .. | 4 | .. | 2 | .. | .. | .. | .. | .. | .. | | | |

EXPLANATION OF TABLE VII.

These patients had never taken quinine before coming into the hospital. The remedy was administered to them in the dose of $1\frac{1}{2}$ grammes per day, during the sweating stage and repeated two days afterwards at the same hour, and so on; in all for seven days. After that the remedy was suspended until the relapse.

EXPLANATION OF TABLE VIII.

The greater number of these patients had already taken quinine very irregularly before coming into the hospital. The treatment was continued within the hospital, but also irregularly, the remedy being given erratically both as regards quantity, hour, and length of treatment.

TABLE VIII.

CASES OF SPRING TERTIAN; QUININE GIVEN IRREGULARLY.

| Days of Ob- serva-tion | No. of Cases | Duration of the Latent Period Calculated in Days | | | | | | | | | | | | | | | | |
|---------------------------|-----------------|--|----|----|----|----|----|----|----|----|----|----|----|----|----|----|--|--|
| | | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | | | |
| 7 | 29 | 12 | 14 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | |
| 9 | 26 | 4 | 7 | 5 | 9 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | |
| 11 | 24 | .. | 7 | 9 | 8 | 6 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | |
| 13 | 20 | 1 | 12 | 4 | 3 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | | |
| 15 | 19 | .. | 1 | 3 | 5 | 2 | 1 | .. | 1 | 2 | .. | .. | .. | .. | .. | .. | | |
| 17 | 10 | .. | 1 | .. | 1 | 1 | 2 | 1 | .. | .. | 1 | .. | .. | .. | .. | .. | | |
| 18 | 19 | .. | .. | 1 | 2 | 3 | 2 | 1 | 1 | .. | 2 | 1 | 1 | 1 | 1 | .. | | |
| 19 | 14 | .. | 1 | 1 | 3 | 2 | .. | .. | 1 | 4 | 2 | .. | .. | .. | .. | .. | | |
| 20 | 16 | .. | 1 | .. | 2 | 5 | 3 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | | |
| 22 | 13 | .. | 1 | .. | 1 | .. | 2 | 4 | 1 | .. | .. | .. | .. | .. | .. | .. | | |
| 25 | 10 | .. | .. | 1 | 1 | 2 | 1 | 1 | 2 | 1 | .. | .. | .. | .. | .. | .. | | |
| 29 | 12 | .. | 1 | .. | 1 | 1 | 3 | 1 | 1 | 1 | .. | 1 | .. | .. | .. | .. | | |
| 30 | 9 | .. | 2 | 1 | .. | 1 | 2 | 1 | .. | 1 | .. | .. | .. | .. | .. | .. | | |
| 31 | 7 | .. | 1 | 1 | .. | 1 | .. | 2 | 1 | .. | .. | .. | .. | .. | .. | .. | | |
| 33 | 10 | .. | .. | 1 | 2 | 1 | 1 | 1 | 1 | .. | .. | .. | .. | .. | .. | .. | | |
| 36 | 11 | .. | 1 | 1 | .. | .. | 1 | 2 | 1 | .. | .. | .. | .. | .. | .. | .. | | |
| 39 | 12 | .. | .. | 2 | 1 | 2 | 1 | 1 | .. | .. | .. | .. | .. | .. | .. | .. | | |
| 40 | 13 | .. | 1 | 1 | 1 | .. | 2 | 1 | 1 | .. | .. | 1 | .. | .. | .. | .. | | |
| 50 | 4 | .. | .. | 1 | 1 | 1 | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | | |
| 60 | 7 | .. | 1 | .. | 1 | .. | 2 | .. | .. | .. | .. | .. | .. | .. | .. | .. | | |
| 70 | 5 | .. | .. | 1 | 1 | 1 | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | | |
| 0 | 2 | .. | .. | 1 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 1 | .. | | |
| Total per day .. | | 17 | 52 | 34 | 43 | 30 | 23 | 18 | 12 | 9 | 5 | 3 | 1 | 1 | 1 | .. | | |

(1) Of 145 cases treated early and systematically, a relapse only occurred in 37 per cent.

(2) Of 301 cases treated systematically, but started somewhat late, there was a relapse in 30 per cent.

(3) Of 1,002 cases with daily quinine there was a relapse in 15 per cent.

(4) Of 50 cases submitted to quinine during the acme, there was a relapse in about 80 per cent.

(5) Of 55 cases given quinine during the sweating stage, there was also a relapse in 80 per cent.

(6) Of 291 cases to whom quinine was given irregularly, there was a relapse in 85 per cent.

(7) Lastly, in 120 cases to whom quinine was not given, there was only one in which a complete cure took place (whence we may regard it as a quite exceptional fact).

(To be continued.)

CARCINOMA AND MALARIA.—Prochnik publishes his observations on carcinoma and its prevalence in malarial countries in the *Weiner Klin. Wochenschrift*, January 30th. He states: that in Java, where he gained his experience, cancer is not at all rare, and that malaria prevails to such an extent that practically every person in Java suffers from malarial infection in some form. He concludes, therefore, that malaria is in no way antagonistic to the evolution of carcinoma. Prochnik even goes so far in the opposite direction as to advance the idea that malaria predisposes to carcinoma of the liver. Hepatic carcinoma, according to Prochnik, is the most common form of malignancy met with in Java.



To illustrate Dr. MITCHELL's article—"A peculiar and undescribed affection of the nose."

(See page 135.)

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THE

Journal of Tropical Medicine

MAY 1, 1902.

PLAGUE IN THE PUNJAB.

THE havoc that plague is causing amongst perhaps the finest race of people in India is from every point of view a calamity. The extent of the epidemic may be judged by the fact that during the months of January and February of this year the deaths from plague in the Punjab numbered 13,466 and 29,493 respectively; and during March they reached the enormous total of 84,949. Taking the population of the Punjab as 20,000,000, the extent and virulence of this epidemic finds no parallel in modern times. The influences that have prevailed to bring about this state of affairs is sought to be attributed to the effects of non-intervention with the customs and wishes of the people, but whether this is scientifically correct is another matter. It would certainly appear that the imposition of stringent regulations as to quarantine, isolation, disinfection, &c., during the years when plague first appeared in

the Punjab, coincided with a distinct limitation of the disease, and that, on the other hand, when these restraints and precautions were removed, plague appeared in a virulent form. Neglecting therefore the possibility of other causes being at work to produce this marked change, it may be useful to trace the history of the disease in the Punjab.

Plague first appeared in the Punjab in 1897, and during the three subsequent years the disease was confined to a comparatively small area, while the mortality was insignificant. During these years it was at one time hoped that plague might be stamped out altogether, but Captain James, who carefully watched the progress of the disease, was of a different opinion. He observed that although the outbreaks were limited, mild, and capable of being controlled, that the area of infection slowly but surely increased in all directions, and that by the winter of 1900-01 a wide tract of country was infected.

During the first years of the outbreak of plague in the Punjab the measures adopted to combat the disease consisted of: (1) Observation of the health of the villages in the infected area; (2) segregation of sick and contacts; (3) restriction of the movements of the population of infected villages by cordons of sentries; (4) disinfection of houses and household property; (5) railway inspection; (6) anti-plague inoculation; (7) surveillance of persons arriving at a healthy village from an infected area—in fact all the hygienic machinery known at the present day was put in force.

Stringent measures such as those indicated above could not be maintained indefinitely, but only when isolated villages were attacked. I was plain that if the disease attained larger dimensions, and if large towns were attacked, these regulations could not possibly be maintained. The very limitation of the outbreak of the disease, however, proved detrimental to the enforcement of sanitary rules, for the people doubted the necessity for such measures. Their attitude, which at first was one of passive obstruction, became in time more active, and culminated in a serious riot at Garshankar.

It is a peculiar feature of the spread of plague in the Punjab that the disease first attacked the small country villages, and even now the larger towns are not seriously affected.

Partly because plague appeared to be limited in virulence and extent, and largely in consonance with the clamouring of the people, the Government consented to remove the restrictions and regulations that had existed for some four years. Coincidentally with this proclamation, and it may be because of it, plague broke out with a severity previously unknown; but had these restrictions not been removed it would have been absolutely impossible to maintain them when plague had attained a firm hold on the population. The necessity for rules and regulations as a means of prophylaxis against plague, and for quite another set of regulations when an epidemic is raging, was never more conclusively proved.

The relative value of the measures to be taken against plague during an epidemic may be enumerated in the following order as to their effectiveness: (1) Prevention of free movement amongst the people; (2) evacuation of infected villages; (3) disinfection of houses, &c.; (4) inoculation.

The Punjabis, since plague has become widespread amongst them, are becoming more amenable to be guided by the authorities. Should that spirit increase, it may be possible to teach them in what direction danger lies and how it can be avoided. Without the intelligent assistance of the people it is impossible to combat a plague epidemic. The people themselves must be taught the importance of excluding persons coming from infected areas, to report suspected illnesses, to see the advantage of segregation of contacts, the benefits of inoculation, disinfection, &c., and to take such public health precautions as are essential. The willingness of the people to aid the Government comes late, but as plague will certainly recur next winter, even should the present epidemic die in the summer as it probably will, the way may be smoothed, so that it may be possible to deal with future outbreaks more effectively.

THE SUBCUTANEOUS INJECTION OF QUININE.

By CHARLES A. BENTLEY, M.B., C.M. Edin.
Borjuli, Tezpur, Assam.

THE discussion that has taken place in the columns of the JOURNAL OF TROPICAL MEDICINE regarding the use of quinine by subcutaneous or intercellular injection has called attention to a subject of great interest to medical men practicing in the Tropics and in all malarial-infected parts of the world. The general tenor of the papers that have already appeared upon the subject seems to indicate that a large number of medical men still regard this method of using quinine with more or less suspicion. It is impossible to deny but that in the experience of large numbers of practitioners there are strong grounds for the belief that solutions of quinine introduced beneath the skin act in a very marked way as predisposing agents for the production of abscesses and obstinate ulceration. I think, however, it will be found that it is not a common occurrence for those who use the methods under discussion as a daily routine practice, to meet with the cases of local gangrene described by so many of the previous correspondents.

In my experience of many hundreds of injections of quinine I have only once had ulceration following upon the injection, and that was in a case when I was obliged by force of circumstances to use an old hypodermic syringe which resisted all one's efforts to render it perfectly aseptic. Since I adopted the hypodermic method of treatment as a routine practice in cases of kala-azar and chronic malarial cachexia with frequent fever, types of malarial infection which are exceedingly common in this part of Assam, and which resist quinine given by the mouth in doses up to 40 grains twice or more in the twenty-four hours, I have not had a single case of abscess or ulceration beyond the one referred to. I have used several of the soluble salts of quinine, but I generally use the bi-hydrochloride, which dissolves easily in its own weight of water. I have injected up to 20 grains of this salt at one time, in a 1 in 2 solution.

I also frequently use a solution of ordinary quinine sulphate made as follows:—

| | |
|---------------------------|----------|
| R. Quinine Sulphate | gr. 160. |
| Acid, Tartaric | gr. 80. |
| Water to | oz. 1. |

This dissolves readily with the aid of heat, and as I always use it either freshly prepared, or reboiled just before use, there is no difficulty about crystallisation. I generally give 20 minims of this to adults, and 10 or 15 to children.

Before giving an injection, the skin between the shoulder blades, or on the buttock, is well scrubbed with 1 in 20 carbolic lotion. Immediately after filling the syringe with the recently-boiled solution I plunge the needle into a bottle of pure carbolic acid and then thrust it deeply into the tissues. After withdrawing the needle and rubbing in the injection I touch the point of entrance with a camel-hair brush or small swab dipped in pure carbolic acid, and finish the business by an application of carbolic oil.

This method is very similar to that mentioned by Dr. Moffat, of Uganda, in the JOURNAL OF TROPICAL

MEDICINE for October 1st, only that I make use of very much more concentrated solutions of quinine than are generally spoken of as being customary. That quinine may be administered with perfect safety in the way I describe is proved by the fact that several of my native assistants now make use of the method I have taught them, and up to the present I have had no case of abscess formation following upon any injection given by them. I recommend the use of an aseptic syringe, all glass, or all metal, or glass and metal, without either rubber, leather or asbestos plungers. They are far more easily cleaned than any other, are more durable, and are always ready for use. I endeavour to keep one syringe for nothing but quinine injections.

As many of these injections, of highly concentrated solutions, have been administered to patients, the subjects of kala-azar, a condition in which there seems to be a marked predisposition to cancrum oris and other acute septic processes, I consider that my experience is, so far, an absolute proof that the subcutaneous injection of quinine is not, *per se*, a cause of abscess formation.

I think it probable, however, that the quinine acts as an inhibitory agent to the phagocytic cells that may be present in the part, and so indirectly cause a lowering of the normal resistance of the tissues to septic processes. It is generally recognised that quinine is not an antiseptic in the strictest sense, and unless the most stringent precautions are taken its administration may be followed by a septic infection occurring in the tissues whose vitality has been lowered by its presence, which would have otherwise been successfully resisted.

No one, I think, who has once adopted the hypodermic administration of quinine for general use, and has witnessed its remarkable efficacy in cases of malarial fever which resist ordinary treatment, would think of going back to other methods of administration.

In many cases here quinine given by the mouth, even in extreme doses, is apparently not absorbed in sufficient amount, as cases of cinchonism are the exception, even where it is being taken in doses of 30 grains at a time. In such conditions hypodermic medication is a necessity for economic as well as therapeutic reasons.

RECENT RESEARCHES CONCERNING THE ETIOLOGY, PROPAGATION, AND PREVENTION OF YELLOW FEVER, BY THE UNITED STATES ARMY COMMISSION.

By WALTER REED, M.D.

Surgeon U.S. Army, President of the Commission.

From the JOURNAL OF HYGIENE, April 1st, 1902.

THE efficient control of the spread of yellow fever is a matter of such vast practical importance, both from the hygienic and commercial point of view—not only for the countries where this disease prevails as an epidemic, but also for those in which, after importation, it may assume epidemic proportions—that it has seemed appropriate to bring together in this paper a summary of the work, thus far accomplished by the

United States Army Commission* on the Island of Cuba, during the years 1900 and 1901, in order that English and Colonial readers who have not, perhaps, had access to the original contributions published in different American journals, may be able to form an intelligent opinion concerning the permanent value of this work. It will also afford opportunity for recording the more recent confirmatory observations made by others concerning the mode of transmission of yellow fever discovered by the Commission, and for calling attention to the results already obtained by the U.S. Army Medical Department in the suppression of this disease, especially in the city of Havana, through the enforcement of sanitary measures based on these later researches.

The American Commission was organised in May, 1900, and began its investigations during the following month (June), being equipped with suitable laboratory facilities for practical work, both at the military garrison of Columbia Barracks, near Quemados, Cuba, and also in the city of Havana. As yellow fever was already prevailing at the time of our arrival in Cuba suitable material for the scientific study of this disease was immediately available.

The Etiology of Yellow Fever.

Before giving the results of our investigations it may be well to recall the situation as regards the etiology of yellow fever at that time. Briefly it may be said that the claims of all investigators for the discovery of the specific agent of yellow fever—since modern bacteriological methods had come into use—had been disproved by the exhaustive observations of Sternberg,¹ published in 1890, except that made by Sanarelli² for a small, motile bacillus isolated by him from the blood drawn during life in two of six cases of yellow fever, and from the blood and organs after death in seven of twelve cases of this disease (58 per cent.) studied at Montevideo and Rio de Janeiro, Brazil. The results obtained, however, by those who had promptly undertaken to investigate Sanarelli's claim for the specific character of *Bacillus icteroides*, seemed to show a lack of agreement such as has never been reported, as far as the writer can recall, in connection with the supposed specific cause of any of the other acute infections. Thus while Achinard and Woodson³ had, during the epidemic of 1897 in New Orleans, La., isolated a bacillus, claimed by them to be identical with *B. icteroides*, from the venous blood in 4 out of 5 cases, and from yellow fever cadavers in 32 out of 39 cases (82 per cent.), Portier,⁴ working in the same city and during the same epidemic, could only obtain this bacillus three times in fifty-one autopsies, and failed to obtain it at all in cultures made from the venous blood during life in 10 cases. Again, while Wasdin and Geddings,⁵ in the city of Havana, were able to cultivate *B. icteroides* from blood withdrawn from the lobe of the ear, "not earlier than the third day of the disease," in 13 of 14 cases (92·8 per cent.), and to find it in 85·7 per cent. of their necrop-

* The members of this Commission were Major Walter Reed, Surgeon, U.S. Army, and Drs. James Carroll, A. Agramonte, and the late Dr. Jesse W. Lazear, Contract Surgeons U.S. Army.

sies, Agramonte,⁸ studying the disease on the Island of Cuba, failed to isolate *B. icteroides* in a single instance from blood drawn from the lobe of the ear in 37 cases or from the blood drawn from a vein at the bend of the elbow in 31 cases, at various stages of the disease. The latter observer, however, reported finding this bacillus at autopsy in 11 of 35 cases (31.4 per cent.). Without going further into detail, we may say that the results obtained by Lutz⁷ and de Lacerda and Ramos⁶ in Brazil, and by Matienzo⁹ in Mexico, were equally conflicting and unsatisfactory.

Under these circumstances it seemed to the members of the Commission of the first importance to give their entire attention to the bacteriological study of the blood of those sick with yellow fever and of the blood and organs of yellow fever cadavers, having especially in view the isolation of *B. icteroides*. We were thus able during June, July and August, to take repeated cultures from the blood during life in 18 cases of yellow fever, adopting the usual method employed in withdrawing blood from a vein at the bend of the elbow, and transferring the blood, at once, in quantities of 0.5 c.c. to each of several tubes containing 10 c.c. of nutritive bouillon which were afterwards incubated at 37° C. for a period of one week. In 7 cases, 4 of which were designated as "mild" yellow fever and 3 as "well-marked" yellow fever, only one culture was made from the blood in each case, viz.: in 2 cases on the first day; in 1 case on the second day; in 3 cases on the third day; and in 1 case on the fourth day. In the remaining 11 cases, diagnosed as "severe" yellow fever, of whom four died, more frequent cultures were taken from the blood, these varying from two to six cultures on as many different days of the disease. In two of the fatal cases, cultures were made each day from the commencement of the attack and including the day on which death occurred.

The negative result of these numerous cultures taken from the blood of cases of yellow fever, as regards the presence of *B. icteroides*, was reported in a "Preliminary Note" presented at the meeting of the American Public Health Association,¹⁰ held in Indianapolis, Indiana, October 22nd—26th, 1900. To these 18 cases we can now add 6 other cases, or a total of 24, from which blood cultures have been made during life with negative results.

The importance of this negative finding as regards the growth of any specific bacterium will be better appreciated when it is seen, as I shall soon have occasion to point out, that yellow fever may be produced in non-immune human beings by the subcutaneous injection of a small quantity (0.5—2 c.c.) of blood withdrawn from the venous circulation of a patient suffering with this disease.

In addition to the results above recorded, the careful study of eleven autopsies was equally barren as to the presence of any particular micro-organism, although the quantity of material with which our tubes were inoculated was greater than is usually made use of at autopsies.

In a word, then, the careful bacteriological study which the Commission had made in cases of yellow fever had given no indications as to the presence of the specific agent of this disease. The same may be

said concerning the result of numerous microscopic examinations of fresh and stained specimens of blood which we had in the meanwhile studied with the view of finding possibly some intracellular or extracellular body. Apparently no body, bacterial or protozoan, which could be brought into view with a $\frac{1}{4}$ Zeiss immersion objective, was present in the blood of these cases.

Although displaced from the order in which the following observations were made, it will be best to present, at this time, the results of the experiments which were later carried out by the Commission on non-immune human beings by means of the subcutaneous injection of blood, withdrawn during the active stage of the disease, as these results bear so directly upon the subject which we are now considering, viz., the etiology of yellow fever.

The only reference that I can find in the literature relative to an attempt to convey yellow fever in this way is cited by Sternberg,¹¹ who states that at Vera Cruz, Mexico, in 1887, he saw Dr. Ruis inject into a non-immune individual a hypodermic syringe of blood drawn from a case of yellow fever on the eighth day of the disease. The result was negative, as was also the result of two other attempts related to him by Ruis.

Our own observations, undertaken for the purpose of ascertaining whether an attack of yellow fever could be induced in a second individual by the injection of a small quantity of blood, embrace experiments made on twelve American soldiers and Spanish immigrants, all non-immune individuals.

These observations may be divided into the following classes:—

(1) Injection of the fresh blood taken from a vein at the bend of the elbow.

(2) Injection of partially defibrinated blood.

(3) Injection of partially defibrinated blood heated for ten minutes at 55° C.

(4) Injection of blood-serum previously diluted with sterilised water and filtered slowly through a Berkefeld laboratory filter.

The following Table, I., gives the results of these several inoculations:—

By an examination of this table it will be seen that of the seven individuals who received subcutaneously the fresh or partially defibrinated blood in quantities of 0.5—2 c.c., six (85.7 per cent.) developed an attack of yellow fever within the usual period of incubation of the disease.

These results are of very great interest as demonstrating that the specific agent of yellow fever is present in the blood, at least during the first, second, and third days of the attack.

Another important point brought out by these experiments was that the blood which conveyed the disease did not contain any bacterium which would grow on our usual laboratory media.

In order to establish this fact, as soon as blood had been injected into the non-immune subject, additional blood was at once withdrawn in considerable quantity and transferred to tubes of nutritive bouillon. In one instance, where 2 c.c. of blood had been drawn into the syringe, 0.5 c.c. of this sufficed, when injected, to produce a severe attack of yellow fever, after seventy-

three hours' incubation, while the remaining 1.5 c.c. transferred immediately to four tubes of bouillon gave no growth, except that from one tube we isolated on the fourth day *Staphylococcus pyogenes citreus*, found by us to be a common skin-contaminating organism in Cuba.

TABLE I.

| No. of Case | Quantity and Material Used | Day of Disease | Date of Inoculation | Result | Date of Attack |
|-------------|--|----------------|---------------------|----------|----------------|
| 1 | 2 cc. fresh blood | Second | Dec. 26, 1900 | Negative | — |
| 2 | 2 cc. „ | „ | Jan. 4, 1901 | Positive | Jan. 8, 1901 |
| 3 | 1.5 cc. „ | First | Jan. 8, 1901 | „ | Jan. 11, 1901 |
| 4 | 0.5 cc. „ | Second | Jan. 22, 1901 | „ | Jan. 24, 1901 |
| 5 | 1 cc. „ | „ | Jan. 25, 1901 | „ | Jan. 28, 1901 |
| 6 | 0.75 cc. partially defibrinated blood | Third | Oct. 15, 1901 | „ | Oct. 20, 1901 |
| 7 | 1.5 cc. partially defibrinated blood heated for 10 minutes at 55° C. | „ | Oct. 15, 1901 | Negative | — |
| 8 | Same as No. 7.. | „ | Oct. 15, 1901 | „ | — |
| 9 | „ „ „ | „ | Oct. 15, 1901 | „ | — |
| 10 | 1.5 cc. of filtered blood serum | „ | Oct. 15, 1901 | Positive | Oct. 19, 1901 |
| 11 | Same as No. 10 | „ | Oct. 15, 1901 | „ | Oct. 19, 1901 |
| 12 | Same as No. 10 | „ | Oct. 15, 1901 | Negative | — |
| | 2 cc. fresh blood | Fourth | Oct. 22, 1901 | Positive | Oct. 23, 1901 |

Table I. further shows that the specific agent contained in the blood is destroyed or attenuated by heating the latter at 55° C. for ten minutes, so that the injection of 1.5 c.c. of this heated blood was harmless (cases 7, 8 and 9), while the injection of 0.75 c.c. of the same blood unheated sufficed to promptly induce an attack of yellow fever in a "control" individual (case 6).

Of not less interest was the fact brought out by these observations that yellow fever can be produced by the injection of a small quantity of bacteria-free serum filtrate, obtained by passing the diluted serum through a Berkefeld laboratory filter (cases 10 and 11), and further that the blood of a case of yellow fever, thus produced, when injected into a third non-immune subject will promptly bring about an attack of this disease (case 12), thus demonstrating that the specific agent of yellow fever can find its way through the pores of a filter which ordinarily serves to prevent the passage of all known bacteria.

I have elsewhere¹ in conjunction with one of my colleagues (Carroll) discussed the facts here presented more at length and will limit myself, therefore, to the remark that these experiments appear to indicate that yellow fever, like the foot and mouth disease of cattle, is caused by a micro-organism so minute in size that it might be designated as ultra-microscopic.

The Propagation of Yellow Fever.

Prior to the time at which the foregoing observations were made the Commission had already turned its entire attention to the possible solution of the problem of the propagation of yellow fever, being induced thereto, not only by the fruitlessness of the investigations made thus far along bacteriological lines, but also by reason of certain facts which seemed to call for a better interpretation than had hitherto been accorded them.

Without entering into details, I may say that, in the first place, the Commission saw, with some surprise, what had so often been noted in the literature, that patients in all stages of yellow fever could be cared for by non-immune nurses without danger of contracting the disease. The non-contagious character of yellow fever was, therefore, hardly to be questioned.

In the second place, it had been observed that patients discharged from the wards during early convalescence could be brought into intimate association with non-immune individuals without thereby establishing fresh foci of the disease. This did not seem to indicate that any specific agent was present in the excreta of the sick.

Again, it had been noted that in certain cases of this disease no growth had been obtained on the ordinary laboratory media, either by frequent cultures from the blood during life or from the blood and organs after death.

Further, in the course of an investigation which the Commission were able to make during the last week of July, 1900, concerning the origin and spread of a small epidemic of yellow fever that had appeared in a military garrison, numbering about 900 men, at Pinar del Rio, Cuba, they had seen that by reason of the false diagnosis of "pernicious malarial fever" which had been given to these cases no disinfection of bedding or clothing had been carried out; and yet there was no indication that this neglect had contributed in the least to the spread of the disease: nor had any harm come to those non-immunes who had slept in the beds vacated by the sick, or washed the supposedly infected garments of those who had recovered or died of this disease.

Putting these various data together, it seemed probable that more progress might be made if attention should be turned to the mode of transmission of yellow fever, especially as our own observations had caused us to seriously doubt the usually accepted belief of the conveyance of this disease by means of *fomites*.

Then, too, the endemic curve of yellow fever in the city of Havana, and its well-known epidemic curve in the United States, appeared to be more intimately associated with and more affected by the rise and fall of the annual temperature curve than was to be seen in any of the acute infections, except malarial fever. The peculiar behaviour of this disease (if I may use the expression) in rapidly spreading in certain localities, when introduced, as contrasted with its failure to propagate itself in other places, where the conditions for its increase were apparently just as favourable, seemed to point in the strongest manner to the neces-

sity for some special agent or intermediate host in the dissemination of its specific cause. If malarial fever—a disease so much affected by temperature conditions—required the agency of a special genus of mosquito for its propagation, as had in recent years been so brilliantly worked out by Ross, Grassi, Bastianelli, Bignami and others, it did not seem unreasonable to suppose that yellow fever—a disease so plainly controlled by seasonal conditions—might also depend on some such agent for its spread. Influenced by this line of reasoning, the Commission began, during the second week of August, 1900, its observations relative to the propagation of yellow fever by means of the bite of a certain species of mosquito—*Stegomyia fasciata*.

The work along this line was carried forward so rapidly that, within thirty days, eleven individuals had been bitten by infected *Stegomyia*, of whom two* developed well-marked attacks of yellow fever within the usual period of incubation, and under such circumstances as to positively exclude, in one case, any other possible source of infection.

Appreciating fully the importance of this discovery and in order to exclude all other possible sources of infection in our future observations, it was now determined to establish a Special Experimental Station where further observations could be made on non-immune human beings, both as to the propagation of yellow fever by means of the bite of the mosquito as well as by exposure to the most intimate contact with infected clothing and bedding, and this under the strictest enforcement of military quarantine. With the approval and assistance of the Military Governor of the Island of Cuba, this Experimental Station was ready for occupancy on November 20th, 1900, and was continuously occupied until March 1st, 1901.

As the results obtained at this station have already been published[†] in full elsewhere, I will here only present a brief account, first of the experiments with fomites and afterwards of those made with infected mosquitoes.

Attempts at Infection by Fomites.

I quote from a paper which the writer presented for the Commission at the meeting of the Pan-American Medical Congress,† held in Havana, Cuba, February 4th-7th, 1901: "For this purpose there was erected at Camp Lazear a small frame house consisting of one room, 14 by 20 feet, and known as 'Building No. 1,' or the 'Infected Clothing and Bedding Building.' The cubic capacity of this house was 2,800 feet. It was tightly sealed within with 'tongued and grooved' boards, and was well battened on the outside. It faced the south and was provided with two small windows, each 26 by 34 inches in size. These windows were both placed on the south side of the building, the purpose being to prevent, as much as possible, any thorough circulation of the air within the house. They were closed by permanent wire-screens of 0.5 mm. mesh. In addition a sliding glass

sash was provided within and heavy wooden shutters without; the latter intended to prevent the entrance of sunlight into the building, as it was not deemed desirable that the disinfecting qualities of sunlight, direct or diffused, should at any time be exerted on the articles of clothing contained within this room. Entrance was effected through a small vestibule, 3 by 5 feet, also placed on the southern side of the house. This vestibule was protected without by a solid door and was divided in its middle by a wire-screen door, swung on spring hinges. The inner entrance was also closed by a second wire-screen door. In this way the passage of mosquitoes into this room was effectually excluded. During the day, and until after sunset, the house was kept securely closed, while by means of a suitable heating apparatus the temperature was raised to 92°–95° F. Precaution was taken at the same time to maintain a sufficient humidity of the atmosphere. The average temperature of this house was thus kept up at 76.2° F. for a period of sixty-three days.

(To be continued.)

CLASSIFICATION OF ANOPHELES OF INDIA.

By W. GLEN LISTON.

Captain I.M.S.

- A. *Palpi unbanded.*
 - I.—A. *Lindesail.*
Tarsi unbanded.
 - II.—A. *Barbirostris.*
Tarsi banded.
 - B. *Palpi banded.*
 - (a) *Palpi with four bands.*
 - I.—A. *Pulcherrimus.*
Tips of hind legs white.
 - II.—A. *Nigerrunus.*
Tips of hind legs black.
 - (b) *Palpi with three bands.*
 - (c) Tips of palpi black.
 - I.—A. *Turkhudi.*
 - (a) Tips of palpi white.
 - I.—Tips of hind legs white.
 - 1. A. *Theoboldi.*
2½ hind tarsal segments white.
Tibiae and femora speckled.
 - 2. A. *Jamesii.*
3½ hind tarsal segments white.
Tarsal joints banded.
 - II.—Tarsal joints banded.
 - 1. A. *Stephensi.*
Two equal large white bands on palpi.
One small band.
Tibiae and femora speckled.
 - 2. A. *Rossii.*
Single large white band on palpi.
Two small bands.
Tibiae and femora unspeckled.
 - III.—Legs unbanded.
 - 1. A. *Culicifacies.*
Only two light spots on wing fringe.
Third longitudinal entirely black.
Five light spots on costa.
 - 2. A. *Listoni.*
Many light interruptions on wing fringe.
Third longitudinal mostly white.
Four light spots on costa.

—*Indian Medical Gazette*, April, 1902.

* One of these cases was that of Dr. James Carroll, Contract Surgeon, U.S.A., a member of the Commission.

† *Loc. cit.*

Current Literature.

HISTOLOGICAL CHARACTERS OF DELHI BOIL.—At a meeting of this Society held on March 12, 1902, Dr. J. H. McLeod exhibited microscopical preparations of Mr. Morris's case of Delhi Boil. A biopsy was made of one of the lesions, a wedge-shaped piece of tissue being excised, which included the purplish-red, raised border and a portion of the centre. Sections were cut and stained by various methods, and the following microscopical appearances were observed:—The initial change appeared to be a cellular infiltration of the corium, in which the cells were collected in foci around the blood-vessels and sweat-coils on the edge of the lesion, but towards the centre they were more or less diffusely distributed, occupying the whole of the corium from the papillary body to the subcutaneous tissue. The cells composing this infiltration consisted of numerous plasma-cells, connective tissue-spindles, a few mast-cells, and a moderate number of leucocytes; here and there multinucleated cells with eight or ten nuclei arranged in the form of a horseshoe and several large homogenised giant-cells were detected. The blood-vessels were not dilated, but there was a definite proliferation of the endothelium. Where the infiltration was densest the fibrous elements had either completely disappeared or had formed a broken-down network between the cells. In the central area the infiltration showed a tendency to break up with the formation of small irregular cavities. There were no signs of caseation, however, but the cells seemed to shrink and crumble, and be replaced by a granular debris. Leucocytes were more numerous in the central area than towards the periphery. Marked changes, which appeared to be of a secondary nature, affected the epidermis. At the edge of the lesion the prickle-cell layer had proliferated irregularly and the prickle-cells were enlarged, the basal layer remaining intact; the transitional layers were thickened and a hyperkeratosis was present, which was evident not only on the surface but also by the formation of horny pearls in the deeper parts of the epidermis. These proliferative changes were much less noticeable in the central area, and here and there in this region the epithelium had actually thinned, and the underlying cellular infiltration had almost reached the surface. A cultivation was made from beneath the horny scales, but gave negative results, and only the *Staphylococcus epidermidis albus* grew. The histological changes exhibited in this case were clearly of the nature of a granuloma with secondary proliferative changes in the epidermis closely resembling those found in tuberculosis verrucosa cutis. The exhibitor hopes to make a further research on the histology and bacteriology of this case, the present sections being shown in order that they might be before the meeting when the case was demonstrated.—*British Journal of Dermatology*, April, 1902.

ERYSIPELAS IN THE NEGRO. REPORT OF A CASE OF THE SO-CALLED SPONTANEOUS TYPE. SUMMARY OF THE LITERATURE ON THE SUBJECT. By Dr. R. P. Scoins.—From the author's case and from the litera-

ture he concludes that the negro possesses no special immunity. The impression prevailing to the contrary is due to the fact that negroes do not apply to the hospitals for treatment, and physicians who have had such cases have not, as a rule, reported them. The onset is attended with enlargement of the cervical glands, sore throat and high fever. The swelling generally begins around the nose or mouth. No cutaneous flush is visible in a very dark negro. Hardened projections can be felt at the periphery of the inflamed area. The blebs are very distinct. Desquamation begins as inflammation subsides, and may be complete in one place while the erysipelatous process is active in another. The general symptoms are those that accompany most of the acute fevers.—*American Medicine*, February 1st, 1902.

MOSQUITOES AND DENGUE.—Dr. Harris Graham, Professor of Pathology in the American College, Beyrout, Syria, publishes in the *Medical Record* of February 8th his investigations concerning the parasite of dengue and the possible transmission of the disease by mosquitoes. According to Dr. Graham, the red blood corpuscles of persons suffering from dengue contain a parasite resembling the plasmodium malaria, but its cycle of production is much longer than that of the malarial parasite. Dr. Graham, in a series of experiments, got positive and negative results according as mosquitoes were and were not allowed opportunity of biting infected persons. He believes he has proved that dengue, failing mosquito inoculation, is not a contagious disease, but that the disease is carried by certain forms of *Culex* from one person to another.

It may be Dr. Graham has made a real discovery, and even reasoning by analogy, the transmission of the disease by mosquitoes is highly probable. At the same time we would take occasion to observe that dengue is but an ill-defined disease. During one outbreak the symptoms are pronouncedly of a malarial type, whilst during another they resemble influenza closely. These considerations, however, do not annul the possibility of dengue being a specific ailment, for the symptoms of many diseases mimic those of other complaints and yet are quite apart.

We await confirmation of Dr. Graham's observation with keen interest.

MALARIAL MIXTURE.—Dr. McIntosh recommends the following:—

| | | | |
|-------------------------------|-----|-----|------------|
| R. Magnesium sulphate | ... | ... | ½ ounce. |
| Solution of ammonium acetate | ... | ... | 1 " |
| Quinine sulphate | ... | ... | 14 grains. |
| Camphor water, enough to make | ... | ... | 12 ounces. |

M.S.—Two tablespoonfuls every four hours.—*New York Medical Journal*, December 14th, 1901.

PLAGUE.

PREVALENCE OF THE DISEASE.

INDIA.—During the three weeks ending March 22nd, 29th, and April 5th, the deaths from plague in India numbered 26,108, 23,286, and 23,834 respectively. These numbers representing the deaths from

plague are more than double those recorded during the corresponding period of 1901. The principal seat of plague at present is the Punjab, where, during the three weeks mentioned above, the deaths from plague amounted to 16,829, 16,675, and 14,871 respectively.

The next most seriously infected area is the Bombay Presidency, where, during the weeks ending March 22nd, 29th, and April 5th, the deaths from plague numbered 3,705, 3,142, and 3,170 respectively. In Bombay during the week ending April 5th, 947 deaths from plague occurred, and in Calcutta 689, both figures showing an increase on those recorded during the week immediately preceding by over 100 cases. In Bengal, exclusive of Calcutta, the returns show that during the three weeks ending April 5th, the deaths from plague amounted to 824, 1777, and 1,256 respectively.

EGYPT.—During the weeks ending April 6th and 13th, the number of plague cases in Egypt were returned as thirteen and twenty-three, and the deaths from the disease during the same periods as nine and thirteen respectively. The Director-General of the Sanitary Department complains that at Karachiels, owing to a native official neglecting to report cases of illness, the disease has spread in the immediate neighbourhood.

In no single town or province of Egypt, however, has plague been allowed to gain a serious hold; and the limitation of the disease to little more than sporadic outbreaks in widely separated districts shows with what vigilance the sanitary authorities are guarding the country from infection.

CAPE OF GOOD HOPE.—After a period of complete cessation from fresh outbreaks of plague lasting some six weeks, it is disappointing to have to report that on April 21st ten cases of plague and five deaths from the disease were reported from Port Elizabeth.

MAURITIUS.—During the weeks ending April 3rd, 10th, 17th, and 24th, the plague returns for Mauritius show the fresh cases as 0, 3, 0, and 0; and the deaths during the same periods as 0, 3, 1, and 0 respectively.

AUSTRALIA.—A telegram from Sydney, April 21st, states that: "Further cases of plague have occurred here, and the disease has appeared among the wallabies at the Zoological Gardens, which in consequence have been closed to the public."

HONG KONG.—During the week ending April 19th seven fresh cases of plague and six deaths from the disease were reported in Hong Kong.

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Archives Russes de Pathologie, de Médec. Clinique et de Bacteriologie.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Brooklyn Medical Journal.

Caducée.
Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Esercito
Hong Kong Telegraph.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
Journal of the American Medical Association.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Medical Record.
Medical Review.
Merck's Archives.
New York Medical Journal.
New York Post-Graduate.
Pacific Medical Journal.
Polyclinic.
Public Health.
Revista de Medicina Tropical.
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The Journal of Tropical Medicine.

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Original Communications.

THE SLEEPING DISEASE (DOENÇA DA SOMNO).

From the Portuguese.

REPORT SENT TO THE PORTUGUESE MINISTER OF MARINE BY THE SCIENTIFIC COMMITTEE SENT TO STUDY THE SLEEPING SICKNESS IN WEST AFRICA, ON FEBRUARY 21st, 1901.

I.

THE members of the Committee appointed to study the Sleeping Disease in the Angola province, arrived at Loanda on May 30th last, having stopped by the way at Principe island where they stayed from the 7th to the 21st of the same month, and then took up their abode on the Sundy estate, property of Senor Jeronymo José Carneiro, some 157 metres above sea-level.

The disease which rages in the island of Principe under the name of the sleeping disease, according to the information which we were able to collect, is of very old standing, and, according to rumour, was imported from Gabiao by slaves, which, previous to 1799, were transported to these latitudes. The intermarriage of these people, natives of the Congo state, with the aboriginal inhabitants, is the source whence the actual population indigenous to the island of Principe is derived.

Previous to 1887 the number of cases of sleeping sickness was small, and it was only after this date that it increased to the serious extent noted during the last three years.

This increase is without doubt owing to the importation of a great number of labourers from the infested regions of the West Coast of Africa, many dying shortly after their arrival and readily transmitting it to their companions, owing to the very bad conditions of maintenance and accommodation on board the vessel.

We do not think it possible, on this estate, that the disease could be attributed to defective sanitary conditions or to the excess of bodily work. Neither have the labourers the opportunity of smoking "liamba," for the plant does not exist here, nor does it constitute an article of importation. Neither can the abuse of spirituous liquors be made the excuse, as they do not form part of the daily rations, a small portion of red wine only being distributed on Sundays. Of the others, the spirit made from the cocoa tree, and distilled on the estate, may be bought by the labourers, but its sale is stringently controlled. Cases of drunkenness are rare.

Of the different negro races, none are immune from attack. Men and women are without distinction victims of the sleeping disease, but it is stated that previous to 3 years of age cases of the disease are not known; as regards the aged nothing could be investigated, as few of them exist on the estate.

Season seems to have no determining influence upon the spread of the disease.

At the Sundy Estate, on which live nearly four hundred labourers, five to ten cases might be in the hospital at one time, collected from no great distance from each other.

INFLUENCE OF CONTAGION.

There are reliable data by which it is suspected that the propagation of the disease is due to contact. We shall give an account of two of the most characteristic evidences of this. In the hospital, a woman suffering from sleeping disease was attended by a servant of the name of Jacinto. It was customary for him to go to the hospital every day, bringing the patient's food, which he ate in common with her, to keep her company. When these people take their food in common they eat it by hand out of the same dish, licking their fingers afterwards.

Jacinto shortly after the death of the patient (twelve or fifteen days) manifested the first symptoms of the

sleeping disease whilst on the voyage to Lisbon, and died in the Hospital of San José.

Three negroes, companions of Jacinto, none of whom visited the hospital, were advised by the Administrator of the island, with the idea of not propagating the disease, that they should abstain from living in intimacy with Jacinto. One of these is at present healthy; the other two were attacked, but with much less intensity; one of the two (which we saw), in spite of all the counsels, continued to frequent Jacinto's house.

The wife and sons of Jacinto, who lived in the same room, never ate in common with the head of the family, according to the custom of their race, and were free from the disease when I saw them a year afterwards.

PERIOD OF INCUBATION AND INVASION.

We were not able to gain any positive information concerning the period of incubation of the disease. The attack may occur suddenly with delirium of a furious type; at other times with homicidal tendencies. Generally there is a previous period of bad health, the lower limbs fail in strength, there is loss of appetite, incapacity for work even in those individuals normally of an active nature, and at last sleepiness, accompanied by rachitic pains and occipital cephalalgia.

THE DURATION OF THE DISEASE.

In respect to the duration of the disease, we have seen cases run a very rapid course; but the evidence, in the absence of *post-mortem* examinations or other means of diagnosis, is not quite satisfactory. Generally the individuals attacked do not die before twenty days, the average duration being two to three months, but cases have been known to live for more than twelve months. On the estate all the individuals attacked by the disease died.

MORTALITY.

With the view of comparing the death-rate of this disease with the general death-rate, and thus to appreciate the conditions which most influence the excessive mortality of this rich and beautiful island, we asked His Excellency the Governor of the district for use of the respective official documents which exist in the various secretary's offices on this particular subject.

These documents, received by the courtesy of the Governor, referred to the year 1900, and in a population of 4,747 souls there were 833 deaths. The proportion being 175.4 per thousand. The mortality was divided amongst the different races: 12 deaths amongst 178 Europeans (67.4 per 1,000); 821 deaths amongst 4,569 individuals of the negro race (179.6 per 1,000); an exceptionally high death-rate.

The total of the deaths include 48 cases from the sleeping disease, all of the negro race, which gives a percentage of 57.6 per 1,000 for the general death-rate, or more strictly 58.4, as there were 12 deaths amongst the whites, which were not due to the sleeping disease.

This percentage we judge to be below the real death-rate, as among other causes of death, we noticed that anæmia, without any explanation, caused 181 deaths,

and some of the deaths in the general statistics attributed to cerebrospinal attacks no doubt belonged to the disease which we are studying.

CLINICAL FEATURES.

Of ten individuals of the negro race attacked by the sleeping disease, to which our attention was drawn, we presented a report based on careful clinical examinations, and on two *post mortems* which we had occasion to perform.

(a) The glandular swelling localised in the mylo-hyoid region and in the front of the neck, was very marked in various cases in the initial period of the disease. The glandular swellings were indolent, hard and movable under the skin and without tendency to suppurate. In one patient the glandular swellings were absent, but he may have had them some time previously.

(b) No localised nervous signs or symptoms were observed in the early stages of the disease; but there was always a marked attitude of indifference, and a notable physical depression in one case, to such a degree that it was absolutely necessary to arouse him energetically and repeatedly to wake him up; but once awake it may be noted that his replies to questions were clear. In respect to muscular movements, nothing was observed similar to paralysis or contraction of the limbs, the patient being possessed of the power of all active movements. Sensibility and the muscular sense seemed intact.

In two cases we had occasion to note the existence of muscular trembling of a more or less permanent character. In one of these, the gait took the form of a series of slight jerks, at one time in one group of muscles, and at another time in another group. Generally we found the Faradic sensibility diminished in the anterior tibial muscles and the biceps of the arms.

SLEEPINESS.

Sleepiness was not a pronounced feature in the early stages of the disease. The soporific state of two patients, who died during our residence in the island of Principe, did not seem to indicate anything very extraordinary, for a similar state is observed, as a general and final episode, in many diseases of a highly debilitating nature. Impotence in men and amenorrhœa in women were constantly observed.

There were other cases under observation which did not show any symptoms out of the common, saving a slight and notable diminution of arterial tension, and in those cases in which it was possible to measure it, by Potani's instrument, oscillated between 10 and 11.

AUTOPSIES.

We made two autopsies, one seventeen hours after death, the other an hour and a quarter after death.

In both cases we found an exudation, more or less of a milky nature under the arachnoid, which showed marked convexity in both hemispheres, invading, in the second body, the upper part of the cerebellum and part of the lateral lobes. This secretion was submitted to microscopic examination and showed the existence of a large quantity of leucocytes and polynuclear leucocytes, with predominance of the former.

On the cerebral vault the milky secretion was diffused, but did not affect the cerebral matter from which the pia mater was easily detached. At other points the effusion went deeper, slightly affecting the pia mater, which became adherent to the visceral layer of the arachnoid. This process of adhesive inflammation, probably of infective origin, existed also in the left cerebral ventricle where the ependyma were adherent a little in front of the fissure of Monro. Neither in the pineal gland nor pituitary body were any abnormal conditions found. The meninges and the spinal medulla were intact, and without any alteration that could be noted by simple ocular observation. In the fluid obtained by lumbar puncture there were found occasionally clots of blood.

The conditions of the improvised laboratory did not permit us to carry on for any length of time very many of these observations, nor could we formulate conclusions other than of a premature nature. But these studies seem to indicate the direction in which we should work in the infectious disease which affects the labourers on the island of Principe.

This Report is signed by the Chief of the Mission, Annibal Bettencourt, June 9th, 1901.

(To be continued.)

THE DURATION OF THE LATENCY OF MALARIA AFTER PRIMARY INFECTION, AS PROVED BY TERTIAN OR QUARTAN PERIODICITY, OR DEMONSTRATION OF THE PARASITE IN THE BLOOD.

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(Translated from the Italian by St. Clair Thomson, M.D. Lond., F.R.C.S.)

(Continued from page 140.)

PART II.

ON RELAPSES AT SHORT INTERVALS.

HAVING thus strengthened the importance of the regular quinine treatment in regard to the proportion of relapses in spring tertian, we come to the examination of the relapses, and we will see the importance of the quinine treatment also upon the duration of the latent period.

We have seen that of 145 cases treated systematically and early, there was a relapse in only 37 per cent.; the number of relapses was greater on the eighteenth day of apyrexia (10), and was less at the tenth day (2). It is to be noted that all these patients (138 in 145) were followed for a long time either in or outside the hospital, and that in those who remained there only a short time (seven to forty days) and who had never had the quick relapse during that interval of time, I have proof that it did not even take place outside, even when they had neglected the quinine treatment.

As we shall see in good time the malarial infection was not, however, exhausted, as there was a late relapse (after about two to six months, of apyrexia) in about one-fourth of the cases.

Of these 37 per cent. who had a quick relapse in the hospital, there was a second relapse only in two, who had had the first relapse on the eighteenth day of apyrexia; when the second relapse was cured in the same way the fever did not return again. The latent period between the first and second relapse was shorter than the primary period of latency, i.e., in one it was fifteen days and in the other sixteen days. In five patients I tried to prolong the treatment beyond the seven days, and in only one of these did we have a relapse on the eighteenth day, after which it relapsed again a second and last time on the sixteenth day of the second apyrexia period. In certain of the patients followed beyond the forty days there was also relapses, but as they were always after a long interval, they come under another heading.

Of the patients treated systematically but starting somewhat late (and we have seen that they amounted to 301), I would say without going into details that, except for the delay in initiating the treatment, these patients were always treated in the same manner as those of the first group.

On the whole there was a relapse in 30 per cent. of the cases, with a maximum of 23 cases on the eighteenth day, and a minimum of 1 on the seventh day; the relapses as a whole kept always increasing from the fifth to the eighteenth day.

In about 200 of the 301 cases followed continuously either in or outside the hospital, we noted that in none of them was there a second relapse at long interval (between two and eight months of apyrexia). Thus, of 9 patients in whom we insisted on the quinine treatment beyond the seventh day (fifteen to twenty-five days), we had a relapse only in 2, and it was late (from the seventeenth to eighteenth day), brief in its duration, irregular, and the fever slight.

Of the 50 patients to whom quinine was given during the febrile acme, there was a relapse in 39 (that is, in about 80 per cent.) between the fifth and the tenth day of apyrexia in continually increasing number, reaching the maximum on the fifth day, and the minimum on the ninth or tenth day of apyrexia.

The second and third relapse at short interval was very frequent in this group of cases. There was no special behaviour in these relapses, and so I do not give details, limiting myself to saying that the period of apyrexia which elapsed between the one and the other was not constant, that a high and regular fever might succeed to a late and more or less irregular fever in a second relapse, and that concomitant symptoms (nausea, vomiting, headache, &c.), varied without any regular type in every relapse. However, with the administration of quinine in the febrile acme we may say that the relapse anyhow comes very soon, and that it is nearly always constant, and notwithstanding that we insisted on the quinine treatment beyond seven days in four of the cases, all of them had relapses two or three times at intervals of five to eleven days.

Of 55 patients treated with quinine during the sweating period, there were relapses in about 80 per cent., in a proportion always less from the sixth day of apyrexia (12 relapses) to the thirteenth day (1 relapse).

The result of administering the quinine is wellnigh the same whether given during the acme or sweating stage. Thus, for example, a relapse appears in four of these patients in whom the treatment was continued beyond the seven days.

With the *regular daily administration of quinine* we showed (as I have already said) the relapse in 55 per cent. between the fifth and the nineteenth day of apyrexia, with a curve at first ascending to the seventh day where it reached the acme (77 relapses), then descending to the tenth or eleventh day (37 cases of relapse), then once more ascending on the twelfth day (42 cases of relapse), and finally descending in a definite way with a minimum of 2 cases on the eighteenth day.

We had then a second relapse and a third in nearly all those who had had the first; besides, in several patients whom I was able to follow for several months both in and outside the hospital, I was able to show a greater or less number of later relapses at short intervals. These appeared as a rule within periods of time which were almost constantly equal for every patient, periods whose duration oscillated between ten and eighteen days.

In two also of these patients there were febrile outbreaks with extraordinary regularity, in the first case for fifteen days, and in the second for eighteen, and so until the succeeding season, in spite of the continued treatment with 1.50 to 2 grammes of quinine per day.

Of the 291 patients in whom the *treatment was carried out altogether irregularly*, the relapses were, as I have said, 85 per cent. with a maximum on the sixth day and a minimum between the sixteenth and eighteenth day of apyrexia.

Besides, in nearly all there was a second and even a third relapse in spite of the treatment. Certain of these patients, although not returning to malarial regions have, however, relapsed at short intervals, and that after a year and a half from the first infection, and also, we repeat once again, in spite of the treatment.

TABLE IX.

SPRING TERTIAN WITHOUT QUININE (IN WHICH ATTACKS DISAPPEARED BY SPONTANEOUS EXHAUSTION).

| No. of Febrile Outbreaks before the Exhaustion of the Fever | No. of Case | Duration of the Latent Period Calculated in Days | | | | | | | | | | | | | | | | |
|---|-------------|--|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|
| | | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 |
| 5 | 2 | 1 | 1 | .. | 2 | .. | .. | .. | .. | .. | .. | 1 | .. | .. | .. | .. | .. | .. |
| 7 | 3 | 1 | .. | 1 | .. | 2 | .. | .. | 1 | .. | .. | .. | 1 | .. | 1 | .. | 1 | .. |
| 4 | 4 | .. | 1 | 2 | 1 | 1 | 2 | 1 | .. | 1 | .. | .. | 1 | .. | 1 | .. | 1 | .. |
| 10 | 5 | 1 | .. | .. | 1 | .. | 2 | 1 | .. | 2 | 1 | .. | .. | 1 | .. | 1 | .. | .. |
| 11 | 6 | .. | .. | 1 | 1 | 1 | .. | 3 | .. | 1 | 2 | 1 | 2 | .. | .. | .. | .. | .. |
| 10 | 7 | 1 | .. | 1 | .. | 2 | 1 | 1 | 1 | .. | 1 | 1 | 1 | .. | 1 | .. | .. | .. |
| 10 | 8 | .. | .. | .. | 1 | 2 | 1 | .. | 2 | 1 | 1 | 1 | 1 | .. | .. | .. | .. | .. |
| 12 | 9 | .. | 1 | 1 | 1 | 3 | 1 | 1 | .. | 1 | 1 | 2 | 1 | .. | .. | .. | .. | .. |
| 10 | 10 | 1 | .. | .. | .. | 1 | .. | 2 | 1 | 1 | 2 | .. | .. | .. | 1 | .. | .. | .. |
| 9 | 11 | .. | 1 | .. | 1 | .. | 1 | .. | 1 | 2 | 1 | .. | .. | .. | 2 | .. | .. | .. |
| 10 | 12 | 1 | .. | .. | 1 | 1 | .. | 2 | 1 | 1 | 1 | 2 | .. | .. | .. | .. | .. | .. |
| 9 | 13 | .. | 1 | .. | .. | .. | 1 | 1 | 1 | 1 | 1 | 1 | .. | 1 | .. | 1 | .. | 1 |
| 6 | 14 | 1 | .. | .. | .. | 2 | .. | 11 | .. | 1 | .. | 1 | .. | .. | .. | .. | .. | .. |
| Total per day | | 7 | 5 | 6 | 9 | 15 | 9 | 23 | 8 | 12 | 11 | 9 | 6 | 1 | 7 | .. | .. | .. |

EXPLANATION OF TABLE IX.

These spring tertians had never taken quinine; in them the fever exhausted itself spontaneously; in all, however, there was a relapse. To this group we must add the single case in which, after a certain number of attacks, the fever spontaneously disappeared, without ever having the febrile manifestations up to the present moment. This patient is still free from fever after twenty-seven months from the subsidence of the tertian.

TABLES X. AND XI. sent by the author were incomplete.

TABLE XII.

PATIENTS WITH SPRING MALARIA TO WHOM QUININE WAS ADMINISTERED IN THE VARIOUS PERIODS OF THE FEVER.

| | Duration of the Latency Calculated in Days | | | | | | | | | | | | | | | | |
|-------|--|---|----|---|----|----|----|----|----|----|----|----|----|----|----|----|----|
| | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 |
| (A) 5 | .. | 2 | 1 | 4 | .. | .. | 1 | .. | .. | 1 | .. | .. | 4 | .. | 1 | 1 | 1 |
| (B) 4 | .. | 3 | .. | 5 | .. | .. | .. | 1 | .. | .. | .. | .. | 6 | 1 | 2 | 1 | 1 |
| (C) 3 | 1 | 2 | .. | 4 | 1 | .. | .. | 1 | .. | .. | 1 | 1 | 3 | .. | 2 | .. | .. |

EXPLANATION OF TABLE XII.

The patients of group (A) were given quinine before the febrile outbreak; the patients of group (B) were given quinine during the febrile acme; and the patients of group (C) were given quinine during the sweating stage.

(2) ON RELAPSE AT SHORT INTERVAL OF SPRING TERTIAN IN PATIENTS WHO HAVE NOT BEEN SUBMITTED TO THE INFLUENCE OF QUININE TREATMENT.

It may be asserted that every spring tertian which is left to itself without the intervention of quinine treatment, after a certain number of more or less regular febrile attacks, will in a certain number of cases present a sudden cessation of these attacks. In other cases the attacks become irregular, or simply increasingly rare, until they finally disappear.

After that we may assert that the relapse is constant after a latency whose duration and character we will study. Only exceptionally does it happen that the attacks do not reappear and that the general condition of the patient improves rapidly; in such the splenic enlargement, the subjective and objective disturbances, and the anæmia diminish and disappear.

I have said exceptionally, since in all of the 120 cases which I was able to follow I was able to note the possibility of such a definite cure in one individual who now, after twenty-seven months of observation, has had no recurrence and in whom I have not been able to detect any signs of malarial infection.

In these tertians, left to themselves, one is able to verify phenomena in regard to which I think it useful to call attention to what is understood by the period of latency. By this name we should denominate the *period of apyrexia intervening between the disappearance of the febrile attacks and their reappearance*.

This distinction seems opportune, because in spite of apyrexia, and in spite of the improvement of the general condition, the patient does not return to his previous condition of health but remains more or less anæmic. From time to time (three to six days) he complains of vague general symptoms—giddiness, weakness, perspirations, anæmia, gastro-intestinal disturbances of brief duration, neuralgia, &c., without fever.

If we examine the blood of a patient during all this period of apyrexia we observe in it almost constantly

the presence of endoglobular parasites which follow a cycle of regular development. As a rule the parasites are scanty, but the phase of development may take place completely. On the other hand they abandon the free forms of "gametes" so very often, that the "gametes" are never solitary but nearly always united two and two, of which one is larger (macrogametes), one is smaller (microgametes); often we can see that the microgametes put forth flagellæ.

With the development in the blood of the endoglobular forms, and particularly at the period of their greatest development, we can note that they correspond with the periods of disturbance above noticed. In these periods we have also the appearance and the increase of reaction of urobilin in the urine (the quantity of urine sometimes diminishes considerably); these urinary phenomena would tend to prove the destruction of the red globules of the blood. Following this subjective and objective phenomena we can establish that their appearance and increase is in relation with the period of greater development of the endoglobular parasites.

The period of latency in them is between the maximum of eighteen and the minimum of five days. The end of this period is announced two to three days previously by the disappearance or by the diminution of the endoglobular parasites in the blood; the free forms present nothing new. After two or three days we have the appearance again of febrile attacks which are each in relation with the development of a tertian generation of endoglobular parasites. The attacks may present themselves from the very beginning as typical tertian; sometimes they may be altogether irregular and atypical. That is to say, we can see repeated exactly what takes place in persons who contract malaria for the first time. This, it is well understood, is when the patient has not been submitted to the influence of any treatment, and when placed in hospital and put under regular regimen he is not appreciably under the influence of any cause likely to excite fever.

Of 319 patients of this group I took note also of the number of relapses, as I wish to see if for a given individual the latent period of one of the various relapses was constant, or if we could find any connection more or less constant which would permit us to foresee the duration of the apyrexia, and in consequence the imminence of the relapse. However, the task presented many difficulties, since for many reasons it is not possible to induce patients to remain long in hospital, nor, when once discharged, is it possible to follow the greater number of them. However, I succeeded in following for more than two relapses 21 out of 319 patients, retaining them in hospital for a variable period of four to nine months. In this way I was able to note that in general the patient relapsed several times, and that the period intervening between one and another relapse was not always the same in every case, but varied within the limits of five to eighteen days, even when the patients were subjected to identical conditions of hygiene and diet.

Hence the number, gravity, and quality of the febrile attacks were not equal in every relapse, for it was possible for a relapse, characterised by a brief

succession of regular attacks, to be followed by another characterised by one long succession of most regular attacks with very high temperature.

Only two patients were followed for quite nine months, the first from October to the following June, and the second from September to May. The first had six relapses and the second five.

I here give in tabular form the relapses, their duration, and the duration of periods of apyrexia:—

First Patient (at the first outbreak of the fever he had six attacks).

PERIOD OF OBSERVATION FROM OCTOBER 1ST TO JUNE 3RD.

| Relap. | Apyr. | Relap. | Apyr. | Relap. | Apyr. | Relap. | Apyr. | Relap. | Apyr. | Relap. |
|-------------|-----------|-------------|-------------|-----------|---------------|-----------|----------|-------------|----------|-----------|
| 17 dys. | 13 dys. | 16 dys. | 16 dys. | 14 dys. | 19 dys. | 15 dys. | 17 dys. | 12 dys. | 10 dys. | 19 dys. |
| Gast. dist. | Regu. lar | Gast. dist. | Irregu. lar | Head-ache | Vertigo vomit | Regu. lar | No dist. | Irregu. lar | No dist. | Regu. lar |

In this patient there were six relapses through the months from October to March: the maximum duration of the relapses were nineteen days to the third and to the sixth relapse, and a minimum of twelve days at the fifth relapse. There was no regular type of local or general disturbance for each apyrexia period; the relapses were sometimes regular, sometimes not. After six relapses the patient remained well some time, but left the hospital not completely cured, there remained anæmia and splenic enlargement. I heard that on returning to the hills he was again seized with attacks four months after the last relapse which occurred in hospital.

In this patient the duration of the apyrexia periods varied from a maximum of eighteen days at the fourth relapse, and a minimum of ten days at the sixth relapse.

Second Patient (he had three febrile attacks at the first invasion of the fever).

PERIOD OF OBSERVATION FROM SEPTEMBER 1ST TO MAY 4TH.

| Apyr. | Relap. | Apyr. | Relap. | Apyr. | Relap. | Apyr. | Relap. | Apyr. | Relap. |
|----------|-----------|---------------|-----------|------------------|-----------|----------|-------------|----------|-------------|
| 15 dys. | 17 dys. | 16 dys. | 15 dys. | 18 dys. | 20 dys. | 17 dys. | 6 dys. | 10 dys. | 9 dys. |
| No dist. | Regu. lar | Vertigo naus. | Regu. lar | Vertigo headache | Regu. lar | No dist. | Irregu. lar | No dist. | Irregu. lar |

In all, five relapses, which followed a long apyrexia of seven months, after which the patient was lost sight of. The maximum duration of the relapses was twenty days in the third relapse; the minimum was six days at the fourth relapse. The apyrexia lasted between eighteen days before the third relapse and ten days before the fifth; there were no special disturbances except vertigo, headache, and on two occasions, nausea. The fever was typical tertian for the first four relapses, only the fifth was irregular.

In these two patients, who were of the same age and occupation, the constitution was equally robust. They were under observation during a sufficiently long time at the same date; they were treated with the same diet and method of life; and they were forbidden to leave their beds.

The relapses in both occurred without special determining causes; they did not suffer cold nor damp, and they had no gastro-intestinal disturbances, except the nausea and vomiting a few hours before the febrile attacks; but these disturbances were not constant;

there were no intercurrent affections during the period of observation. The observations were identical in the 19 other patients in whom we were able to study more than two relapses, hence I do not think it necessary to discuss them at length. I would only say that I was able to note in these 21 patients that the relapses occurred more frequently during the spring than during the winter months. I verified this in all the 120 patients of this group; in fact, many of them who had no fever in the autumn, relapsed regularly in the spring. The maximum of relapses was from March to May; moreover, these observations of mine coincide with all authors who have studied malaria. Recent observations carried out most scrupulously have proved this in a definite manner.¹

(3) SUMMARY.

Summarising the results obtained from the tables and the facts cited, we may conclude that:—

(A) In patients submitted systematically to quinine at the first outbreak of fever:—

(1) The relapse with short interval only occurs in 37 per cent. of the cases, that is to say, they offer the lowest proportion of relapses.

(2) The relapse at short interval is only a single one, since in only 2 cases were there two relapses, that is, it would occur in 1.5 per cent.; in such a case the relapse is shorter.

(3) The relapse at short interval is more frequent towards the nineteenth day, occurring on that day in about one-quarter of the cases.

(4) By prolonging the treatment beyond the seven days, the relapse occurs all the same (in each case the fever relapsed on the eighteenth day for the first time, and on the sixteenth day for the second), hence one may probably conclude that the prolonged quinine treatment has no influence, neither preventing nor retarding the relapse in spring tertian.

(B) In individuals systematically treated after they have already had some febrile attack, analogous in other conditions to those mentioned under (A):—

(1) The relapse at short interval only occurs in 30 per cent. of the cases, and the patients treated in this way present the lowest proportion of relapses.

(2) The relapse is more frequent between the fifth and the eighteenth day, and reaches its maximum on the eighteenth day (it occurred in 23 cases).

(3) There is no second relapse, or at least very rarely, opportunity never having arisen to us to study any such case.

(4) The prolonged quinine treatment has no influence, neither preventing nor retarding the relapse.²

(C) With the regular daily administration of quinine:—

(1) We have relapse in 15 per cent., with two maxima, one at the seventh and the other at the twelfth day, the minimum at the eighteenth day.

(2) The second and third relapses are the most frequent.

(3) In the case of more than two relapses the

intermediate apyrexial periods were often of equal duration for every patient.

(4) By insisting on the quinine treatment these data do not vary.

(D) With the quinine administration during the acme and during the sweating, the relapse occurred:—

(1) In 39 cases in 50, that is, in nearly 80 per cent., that is these patients furnish a proportion of relapses far higher than the preceding categories.

(2) The relapse at short interval appeared even earlier than with the other methods of quinine administration, reaching the maximum proportion on the fifth and sixth day, and the minimum on the ninth and thirteenth day.

(3) The second and third relapses at brief interval, were the most frequent.

(4) By insisting on the quinine treatment beyond five days the relapse is equally frequent, and so indeed are the second and third relapses.

(E) In the patients treated in an irregular way:—

(1) The relapse is more frequent in the early days than towards the eighteenth day, the period of latency being maintained in a duration of five to eighteen days.

(2) The relapse at short interval occurs in 85 per cent., that is, these patients present a proportion of relapse still higher.

(3) More relapses occur in the same individual.

(4) The insistence on the treatment does not influence the recurrence more or less of the relapses.

(5) These patients, like some of those treated with quinine in the acme and during the sweating, and like those treated with quinine regularly every day, can have relapses at short interval, atypical, for months, and sometimes for years.

(F) In the spring tertians without quinine:—

(1) The spring tertian can exhaust itself definitely but only very rarely, in the greater number it remains latent; this period of short latency is of the minimum duration of five days, and maximum of eighteen days; the maximum of relapses occurs between the ninth and eleventh day of apyrexia from the spontaneous cessation of febrile attacks.

(2) We can have disturbances more or less vague, not associated with fever, between the third and sixth day of apyrexia, disturbances which for other reasons (examination of blood, urinary examination) can be perhaps attributed to abortive febrile attacks. All this consequently permits us to suppose that as a rule the period of latency between the various relapses may be brief, and also that if we would consider as abortive attacks the local and general disturbances, such a period can often arrive only at the three days. However, given the constant fact that the examination of the blood is always negative, two or three days before the outburst of fever, and only then while during the so-called disturbances that does not occur, we may limit ourselves to considering as symptoms of relapse only those which are associated with a rise of fever, and limit the period of latency between the fifth and eighteenth days; that is, however, remembering that the relapse is always easier between the ninth and the eleventh day.

(3) These patients furnish the greatest proportion

¹ Celli, *loc. cit.*

² Probably when the quinine treatment is applied after some fever, the remedy acts upon a generation of parasites, probably less resisting, and hence such a systematic quinine administration a little retarded gives the best therapeutic results.

of relapses to the relapse at short interval. In these patients the relapse is repeated after a period of time which may not be the same in each one although in the same individual, but always manifesting itself in the given period of five to eighteen days.

(4) The relapse at short interval is more frequent in the spring and chiefly between March and May, but without the period of latency indicating any influence in its duration.¹

CHAPTER 2.—QUARTAN FEVER.

I have placed before all observations those carried out on spring tertian in relation to the period of latency in the relapses at short interval. In speaking of quartan and of malignant tertian, I will refer briefly to many of the factors minutely described under spring tertian. In the exposition of the facts relating to quartan I will maintain the same arrangement that I have followed so far; that is, following the conception of the quinine administration, I will demonstrate the results, adding one by one all the observations in relation to the provocative changes of the relapse, as I have done with the spring tertian.

As we have seen previously, the cases of quartan I have studied amount to 155, distributed as in Table No 1.

Of these only 55 were of the triple form of quartan and 100 of the double form.

(1) QUARTAN SUBMITTED TO QUININE TREATMENT.

All the patients with quartan treated with quinine, when under good hygienic conditions, remained immune to relapse at short intervals so long as they remained in these conditions, and there was no difference in them with reference to the method in which the quinine was administered. I do not reproduce, however, the figures which indicate the number of these patients divided in groups, as I did for the spring tertians; I limit myself only to saying that of each group some were followed for three or four months without presenting any pathological manifestations. It would result, however, from my observations that the quartans submitted to quinine treatment, if they maintained themselves in good conditions of life, did not relapse at short intervals. However, that is always in patients treated in hospital, or who, in their own house, can maintain the same care as at the hospital. I can assert that there was no difference of any sort in reference to age or sex.

(To be continued.)

METHYLENE BLUE IN MALARIA.—A de Blusi, in the *Gaz. de Ospedali* (Milan) of March 16th and 23rd, 1902, states that he treated 100 cases of malaria with methylene blue in doses of from 20 cg. to 2 grms. in the twenty-four hours. He claims cures in sixty-two of the cases recorded by this treatment. Blusi warns practitioners against administering methylene blue during pregnancy.

¹ The intervention of other factors which we call accidental, may abbreviate in particular cases the duration of the period of latency.

PREVAILING DISEASES IN BRITISH HONDURAS.—Like all other Central African countries, British Honduras has its share of *malaria*, the most prevalent disease. The lowlands along the coast are the breeding-places of the plasmodium. Many of the residents, foreign and native, who have lived for several years in malarial districts show plain indications of chronic malarial poisoning. The pernicious form and that complicated by hæmaturia are occasionally met; the quotidian type is the most common.

Yellow fever has never had its origin here; the several epidemics which have scourged the country at different times could always be traced to cases coming from Cuba, Port-Limon, or South America, more especially Colon.

Tuberculosis is prevalent among the Indians, Caribs, and half-breeds. The lungs are most frequently the primary seat of the disease, but glandular, bone and joint tuberculosis are by no means rare. The mixture of races has here, as elsewhere, been a potent element in the rapid dissemination of the disease.

Pneumonia and *rheumatism* are most common during the rainy season.

Dysentery and *diarrhæa* occur at all times of the year, and to them must be attributed the frightful infantile mortality. The frequency of intestinal disorders can be accounted for by improper food, inadequate clothing, exposure to rain, prolonged heat, and high atmospheric humidity. The maximum shade temperature is about 90° F., minimum, 56° F.

Whooping cough invaded the colony in October, 1898, has continued to prevail from time to time, and has contributed largely to the mortality of infants, and raised the death-rate from respiratory diseases.

Typhoid fever is a very rare disease here as elsewhere in Central America. The rarity of the disease in Belize may be attributed to the water supply, as this is on the "separate" system, that is, to a considerable extent each house has its supply from a separate vat, the water being rain-water collected from the roof. A few years ago there were four cases of *leprosy* in British Honduras; at present only one.

Anchylostoma was reported for the first time in the colony in 1898. Since that time it has been frequently observed. I was given an opportunity to examine two cases in the Public Hospital. In both instances the disease was characterised clinically by puffiness of the face and pronounced anæmia. In Corosal "it was observed in selected cases that this worm was almost invariably present, associated with *ascaris lumbricoides* and *oxyuris vermicularis*"; and in Orange Walk the medical officer reports: "It is frequently encountered, and is responsible for much of the anæmia prevalent among the Indian population" (Eyles).

Cases of *beri-beri* are occasionally seen, both imported and indigenous. One case of this disease, the paralytic form, is in the public hospital at the present time. The patient is a woman of middle age. The muscles principally involved are the extensors of the hand and fingers.—N. Senn, M.D., *Jour. Amer. Med. Assoc.*, April 26th, 1902.

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THE

Journal of Tropical Medicine

MAY 15, 1902.

HOSPITAL ADMINISTRATION IN THE CROWN COLONIES AND PROTECTORATES.

IN our Colonies and Protectorates we have many Hospitals, some of them of great size, excellently administered and equipped. For the most part they are under the management of medical men who not only treat the sick but are responsible for the administration and organisation of these large establishments. In our civil hospitals in Britain medical men are merely the surgeons and physicians; they are not concerned in the working of the hospital, nor, in fact, are they expected or allowed to take part in so doing. The administration, organisation and equipment, even the superintendence of the nursing staff is in the hands of a lay committee representing the subscribers and donors to the Hospital Funds.

One of the reasons why medical men in Britain are thus shut out from the management of the

hospitals to which they are attached, is no doubt owing to the fact that they have no training in hospital management, and do not interest themselves in the matter. It appears incongruous, but it is so, that the medical staff of a purely medical institution should be under the control of laymen, and what would seem surely part of a medical man's duties, namely, the working of our hospitals, is conducted by a body of men who know nothing of medicine or its actual requirements. The position of the staff is sometimes—but only sometimes—so interfered with, that the lay committee may actually reject for a surgical or medical appointment, a candidate selected and recommended by the medical and surgical staff. The cause of this anomaly in civil hospitals in Britain is no doubt owing to the fact that the public supply the money, and they consider, and justly too, that they have a right to know how the moneys subscribed by the public are disposed of.

In our civil hospitals connected with the Colonial Medical Service it is the principal medical officer who is responsible for the organisation and administration of the hospital for the most part. He is almost in the same position as the naval or military medical officers in charge of hospitals, being at once the medical head and the administrator of the establishment with which he is connected.

One naturally asks what training in hospital administration have the members of the Colonial Medical Service when they join. In the Army and Navy this branch of service is regarded as one of the most important, and it is necessary to have theoretical and practical training, and to pass examinations of a searching character in hospital administration before taking charge of a hospital. In no branch of work have the medical officers of the Army gained greater credit, in the opinion of the civil medical men who worked with them or under them in South Africa, than in their power to administer and organise. Even quite junior officers of the R.A.M.C. astonished all the civil consultant surgeons and physicians attached to hospitals at home, in the efficient and "business-like" way they provided for and super-

intended the camps, field hospitals, &c., during the time of war. Many of the older and more experienced civil surgeons whilst in South Africa resented their hospitals being under the control of a junior Captain or a Lieutenant of the R.A.M.C.; but they soon saw that this officer, junior though he was, proved to be their superior in knowledge of how to administer a hospital, and they were bound to humbly submit to the *régime*, owing to their own lack of acquaintance with the subject. The handle the military officer had was that he had been practised, trained and examined in how to administer.

In the Civil Hospitals in our Colonies the medical officer gathers, as years advance, experience which fits him to be in charge of a hospital when the time comes for him to take over the duties. This experience, however, gives him only a knowledge of the local requirements; a useful and necessary knowledge no doubt, but it is a knowledge gained after the fashion of a housewife—useful but local, and may be good and economic or bad and extravagant. This should not be; hospital administration ought to form part of every medical man's training, but especially of officers about to join the Colonial Medical Service, or about to become members of that most useful and commendable section of our profession, medical missionaries. Mission hospitals are, perhaps, more completely under the management and control of medical men than are any other hospitals, be they civil, naval or military, at home or abroad, and there are no more economically managed hospitals in the world; nor any in which so much good is got out of a small amount of money. Doctors can become "business" like, and are to be implicitly relied upon as administrators, but it is not fair to ask them to assume this great responsibility without an education in hospital organisation. Such training would be a great boon to the doctors themselves, to the service or society to which they belong, and for the economy and welfare of the hospital over which they have control.

The London School of Tropical Medicine could not do better than set the example, and institute a course of technical training in hospital adminis-

tration. By so doing they would bestow direct benefit upon our civil and missionary hospitals in our colonies and possessions, and indirectly, perhaps, stimulate other educational bodies to follow their example, so that medical men might be trained in a branch of their work which they have neglected, but which surely is peculiarly their own.

NAVAL MEDICAL SERVICE.

SIR,—I gather from your letter in the *British Medical Journal* that you invite criticism on the Naval Medical Service.

(1) As to your remarks regarding marriage of naval surgeons, it is financial suicide; they have to keep up the full expenses of a ward-room mess in addition to wife's household. In this and every other matter the R.A.M.C. compares favourably.

(2) The pay of naval surgeon, whether at home, up the Persian Gulf, or West Africa, is the same; here, again, the R.A.M.C. scores in getting Indian and Colonial allowances.

(3) There is no *charge* pay for naval surgeons, though it is given to every other grade in Navy—executive, engineer or paymaster.

(4) The naval surgeon is only given relative (with but after) rank of lieutenant for *twenty* years. This is practically the whole service of the bulk of men who enter. Imagine a man who may be nearly 48 years of age ranking with lieutenants for choice of cabin, &c.

(5) Numbers of naval surgeons never join a hospital during their service. How can a man keep *au courant* with medicine and surgery?

(6) Boiled down, the new warrant gives 3s. a day extra pay, and nothing else, and the half-pay is left as before. And the day a man ceases to be borne on a ship half-pay begins.

As an improvement in their position, naval surgeons have for some years asked:—

(1) For promotion to staff surgeon in eight years. For promotion to fleet surgeon in sixteen years.

(2) For charge pay at same rates as is granted to other branches (executive, engineers and paymasters).

(3) That after each commission a surgeon shall pass not less than three months at one of the large naval hospitals (Haslar, Plymouth, Chatham), or at a metropolitan hospital, before proceeding on a fresh cruise.

It is the general opinion that, unless these very moderate concessions are granted, young medical men will do better in the R.A.M.C. or out of the Services altogether.

Yours, &c.,

R. N.

A COLONIAL MEDICAL SERVICE.

April 3rd, 1902.

SIR,—We, the undersigned Medical Officers belonging to the Colonial Medical Service, having read with much interest a leading article in the *JOURNAL OF TROPICAL MEDICINE* for December 2nd, 1901, on the

subject of a proposed Medical Service for the Colonies and Protectorates, and having voted that medical men in the Crown Colonies and Protectorates are invited to express their opinions on the scheme formulated by the Crown Colonies and Protectorates Medical Service Committee, are glad to have an opportunity of expressing our entire sympathy and accord with the proposal that a Colonial Medical Service be formed. The details of the scheme, as set forth by the Committee, are in accordance with our views.

With regard to pay, we are of opinion that regular increases of salary should be given according to seniority, and that the actual salaries should not vary in the different Colonies and Protectorates, but that especial allowances should be given to those serving in places known to be unhealthy. For this purpose the various Colonies and Protectorates should be graded according to climate.

The question of leave has not been dealt with in the draft scheme. The local leave regulations, under which no leave beyond three months' full pay leave can be taken until the completion of six years' service, are in our opinion unsuitable, inasmuch as there is no doubt but that six years is too long for a European to remain in a purely tropical country without a change of climate. We would recommend that eight months' full pay leave be granted in respect of every four years' service in a healthy tropical climate.

With regard to pensions and gratuities we entirely agree with the suggestions of the Committee. The local pension regulations, under which no pension can be taken until an officer has served for thirty-five years or is fifty-five years of age, practically means that few officers will ever live to earn a pension.

We have the honour to be, Sir,

Your most obedient servants,

[Signed by four Officers of the
Colonial Medical Service.]

The Editor,

The Journal of Tropical Medicine.

SLEEPING SICKNESS IN UGANDA.

APPOINTMENT OF A SCIENTIFIC EXPEDITION.

UNDER the auspices of the London School of Tropical Medicine an expedition is to start early in June to study and report up the development of sleeping sickness in districts in and around Uganda. The members of the expedition consist of: Dr. Castellani, bacteriologist; Dr. Christy, epidemiologist; and Dr. George Lord, parasitologist. Dr. Lord is in charge of the expedition. The advent of sleeping sickness in this region of Africa is quite recent, as we have mentioned several times in previous issues of the Journal, and the serious nature of the disease may be gathered from the fact that in the Busogo district alone 20,000 persons are reported to have died recently from sleeping sickness, and in the district of Chugwe the mortality from the same cause is reported to be heavy. Until a year or two ago sleeping sickness was regarded as a curiosity, but seeing that the disease and its "accompanying" parasite, the *filaria perstans*, have both crossed from the water-shed of the Congo

to that of the Upper Nile, and that the disease is showing a virulence hitherto uncredited, it would appear as if sleeping sickness were about to become a veritable scourge. The funds for the expedition have been provided by the Royal Society and the Foreign Office.

RECENT RESEARCHES CONCERNING THE ETIOLOGY, PROPAGATION, AND PREVENTION OF YELLOW FEVER, BY THE UNITED STATES ARMY COMMISSION.

By WALTER REED, M.D.

Surgeon U.S. Army, President of the Commission.

From the JOURNAL OF HYGIENE, April 1st, 1902.

(Continued from page 146.)

"November 30th, 1900, the building now being ready for occupancy, three large boxes filled with sheets, pillow-cases, blankets, &c., contaminated by contact with cases of yellow fever and their discharges were received and placed therein. The majority of the articles had been taken from the beds of patients sick with yellow fever at Las Animas Hospital, Havana, or at Columbia Barracks. Many of them had been purposely soiled with a liberal quantity of black vomit, urine, and faecal matter. A dirty 'comfortable' and a much-soiled pair of blankets, removed from the bed of a patient sick with yellow fever in the town of Quemados, were contained in one of these boxes. The same day, at 6 p.m., Dr. R. P. Cooke, Acting Assistant Surgeon, U.S.A., and two privates of the Hospital Corps, all non-immune young Americans, entered this building and deliberately unpacked these boxes, which had been tightly closed and locked for a period of two weeks. They were careful at the same time to give each article a thorough handling and shaking, in order to disseminate through the air of the room the specific agent of yellow fever, if contained in these fomites. These soiled sheets, pillow-cases and blankets were used in preparing the beds in which the members of the Hospital Corps slept. Various soiled articles were hung around the room and placed about the bed occupied by Dr. Cooke.

"From this date until December 19th, 1900, a period of twenty days, this room was occupied each night by these three non-immunes. Each morning the various soiled articles were carefully repacked in the aforesaid boxes, and at night again unpacked and distributed about the room. During the day the residents of this house were permitted to occupy a tent pitched in the immediate vicinity, but were kept in strict quarantine.

"December 19th those three non-immunes were placed in quarantine for five days and then given the liberty of the camp. All had remained in perfect health, notwithstanding their stay of twenty nights amid such unwholesome surroundings.

"During the week December 20th—27th the following articles were also placed in this house, viz., pajamas suits, 1; under-shirts, 2; night-shirts, 4; pillow-slips, 4; sheets, 6; blankets, 5; pillows, 2; mattresses, 1.

These articles had been removed from the persons and beds of four patients sick with yellow fever and were very soiled, as any change of clothing or bed-linen during their attacks had been purposely avoided, the object being to obtain articles as thoroughly contaminated as possible.

"From December 21st, 1900, till January 10th, 1901, this building was again occupied by two non-immune young Americans, under the same conditions as the preceding occupants, except that these men *slept every night in the very garments worn by yellow fever patients throughout their entire attacks*, besides making use exclusively of their much-soiled pillow-slips, sheets and blankets. At the end of twenty-one nights of such intimate contact with these *fomites*, they also went into quarantine, from which they were released five days later in perfect health.

"From January 11th till January 31st, a period of twenty days, 'Building No. 1' continued to be occupied by two other non-immune Americans, who,

individuals who were lodged in tents in a separate part of the camp, were being subjected, with their full consent, to the bites of mosquitoes which had previously fed on the blood of cases of yellow fever occurring in the city of Havana. Thus, during the period from December 5th, 1900, to February 7th, 1901, we had subjected to this method of infection twelve non-immune subjects, who had previously passed their full record of quarantine in this camp. Of these ten, or 83.3 per cent., experienced attacks of yellow fever and always within the period of incubation of this disease.

The following Table II. gives the necessary data concerning these observations.

The positive results obtained, therefore, by this mode of infection, stand in striking contrast to the negative experiments made with *fomites*. Indeed, cases 8 and 9 of Table II. had each slept twenty-one nights in the garments of yellow fever patients while occupants of Building No. 1. As they had remained

TABLE II.

| No. of Case | Days in Quarantine | INOCULATION | | Method of Inoculation | Period of Incubation in Hours | Result | Order of Occurrence | Date of Occurrence |
|-------------|--------------------|-------------|-------------------|-----------------------|-------------------------------|----------|---------------------|--------------------|
| | | Hour | Date | | | | | |
| 1 | 15 | 2 p.m. | December 5, 1900 | Mosquito | 81½ | Positive | 1 | December 8, 1900. |
| 2 | 9 | 4 p.m. | " 8 " | " | 137 | " | 3 | " 13 " |
| 3 | 19 | 10.30 a.m. | " 9 " | " | 83½ | " | 2 | " 12 " |
| 4 | 21 | 4.30 p.m. | " 11 " | " | 91½ | " | 4 | " 15 " |
| 5 | 32 | 12 noon | " 21 " | " | 95 | " | 5 | " 25 " |
| 6 | 31 | 10 a.m. | January 8, 1901 | " | — | Negative | 6 | — |
| 7 | 22 | 11 a.m. | December 30, 1900 | " | 94½ | Positive | 7 | January 3, 1901. |
| 8 | 69 | 8.30 p.m. | January 19, 1901 | " | 95½ | " | 8 | " 23 " |
| 9 | 74 | 10.30 a.m. | " 25 " | " | — | Negative | 9 | " |
| 10 | 6 | 9.30 a.m. | " 31 " | " | 74½ | Positive | 10 | February 3, 1901. |
| 11 | 78 | 11 a.m. | February 6 " | " | 78 | " | 11 | " 9 " |
| 12 | 25 | 2 p.m. | " 7 " | " | 70 | " | 12 | " 10 " |

like those who preceded them, have slept every night in the beds formerly occupied by yellow fever patients and in the night-shirts used by these patients throughout the attack without change. In addition, during the last fourteen nights of their occupancy of this house they had slept each night with their pillows covered with towels that had been thoroughly soiled with the blood drawn from both the general and capillary circulation, on the first day of the disease, in the case of a well-marked attack of yellow fever. Notwithstanding this trying ordeal these men have continued to remain in perfect health.

"The attempt which we have therefore made to infect 'Building No. 1' and its seven non-immune occupants, during a period of sixty-three nights, has proved an absolute failure."

Infection by Mosquitoes.

While the experiments with *fomites* were being carried out in "Building No. 1," certain non-immune

in perfect health at Camp Lazear for yet thirty days longer, they were at the expiration of this time bitten by infected mosquitoes solely for the purpose of testing their immunity and with the result that an attack of yellow fever promptly followed in each case.

It should be borne in mind, also, that of the non-immune residents at Camp Lazear, while all lived under the same hygienic conditions, only those individuals developed yellow fever who were purposely bitten by contaminated mosquitoes, or injected with the blood of those sick with this disease. Moreover, the precision with which the infection of the individual followed the bite of the mosquito left nothing to be desired in order to fulfil the requirements of a scientific experiment.

Case 5 of Table II. is of especial interest, when taken in connection with the failure to induce the disease by contact with *fomites*.

This individual, having been quarantined for thirty-two days at Camp Lazear, volunteered to enter a

newly-erected building in which fifteen contaminated mosquitoes had just been freed. His first visit was at noon, December 21st, 1901, and the length of his stay thirty minutes. At 4.30 p.m., the same day, he again entered this building and remained twenty minutes. The following day, at 4.30 p.m., he, for the third time, visited this room and remained twenty minutes. During each of these visits he was bitten by mosquitoes. He did not enter the building again, nor was he exposed to any other source of infection. Nevertheless, at the expiration of three days and twenty-three hours, or at 6 a.m., December 25th, 1900, he was suddenly seized with an attack of yellow fever, which proved to be severe in character. That the infection was occasioned by the bites of contaminated mosquitoes was plainly shown by the immunity from the disease enjoyed by two non-immunes "controls," who, protected only by a wire-screen partition, had been present at each of the subject's visits and who, under the same conditions of security against the bites of the infected mosquitoes, continued to sleep in, and breathe the common atmosphere of this room for yet eighteen nights.

To the positive cases contained in Table II, which were produced at Camp Lazear, we are now able to add four other cases of yellow fever occasioned by the bites of infected mosquitoes, thus making a total of 14 cases, in each of which happily recovery followed.

A very important point brought out by these observations is that an interval of about twelve days or more after contamination appears to be necessary before the infected *Stegomyia* is capable of conveying the disease to a susceptible individual. Repeated experiments made with insects which had bitten yellow fever patients two to ten days previously were always negative, although these same insects were proven capable of conveying the disease after having been kept until seventeen to twenty-four days had elapsed. Our observations¹⁴ further demonstrate that mosquitoes that have been kept for periods varying from thirty-nine to fifty-seven days after contamination are still capable of conveying the disease, and further that infected *Stegomyia* may survive for a period of at least seventy-one days. This will explain how the contagion of yellow fever may cling to a building, although it has been vacated for a period of two or more months.

Bearing in mind that the observations made by means of blood injections (Table I.) were only undertaken *after* we had succeeded in demonstrating that the disease could be conveyed by the bites of the infected *Stegomyia*, it will be seen that our study of the method of propagation of yellow fever at Camp Lazear sufficed to prove very definitely that, while the natural mode of transmission of this disease is through the bites of infected mosquitoes, yellow fever may also be conveyed, like malarial fever, by the injection of a small quantity of blood taken from the veins of an individual suffering with this disease.

Per contra, our observations show that, notwithstanding the common belief in this mode of transmission, yellow fever cannot be induced in the non-immune individual even by the most intimate contact with contaminated articles of clothing and bedding.

Although the investigations made at Camp Lazear were only concluded one year ago, already confirmatory evidence of the strongest character has been furnished in a series of experiments carried out by Guit  ras,¹⁵ at the Inoculation Station of the Sanitary Department of Havana.

I may be pardoned for quoting the paragraph with which Guit  ras begins his contribution. He says: "The favourable results obtained by the United States Army Commission in their experiments with yellow fever, the continued series of mild cases resulting from these experiments without a death, suggested very naturally the continuation of their work on a larger scale; not with a view to control or confirm the conclusions of the Commission, for anyone who had followed their work with unprejudiced attention must have concluded that their solution of the problem of the etiology of yellow fever was final; but rather in the hope of propagating the disease in a controllable form, and securing amongst the recently arrived immigrants immunisation, with the minimum amount of danger to themselves and the community."

Of a total of 42 individuals inoculated by Guit  ras 25 were rejected by him by reason of having been bitten by insects that had been applied to cases of fever about which the diagnosis was in doubt. The following table, therefore, only includes 17 persons who were bitten by *Stegomyia* that had previously fed on unmistakable cases of yellow fever at intervals of fourteen to thirty-six days before being applied to the non-immune subject.

TABLE III.

| No. of Case | Date of Inoculation | Mode of Inoculation | Result | Period of Incubation |
|-------------|---------------------|---------------------|----------|----------------------|
| 1 | Feb. 23, 1901 .. | Mosquito | Positive | 3 days, 10 hours. |
| 2 | Aug. 4 " .. | " | Negative | — |
| 3 | " 4 " .. | " | " | — |
| 4 | " 7 " .. | " | " | — |
| 5 | " 8 " .. | " | Positive | 4 days, 5 hours. |
| 6 | " 8 " .. | " | " | 3 " 3 " |
| 7 | " 7 " .. | " | Negative | — |
| 8 | " 9 " .. | " | Positive | 5 days, 3 hours. |
| 9 | " 10 " .. | " | Negative | — |
| 10 | " 10 " .. | " | " | — |
| 11 | " 10 " .. | " | " | — |
| 12 | " 13 " .. | " | Positive | 3 days, 19 hours. |
| 13 | " 13 " .. | " | Negative | — |
| 14 | " 14 " .. | " | Positive | 3 days, 21 hours. |
| 15 | " 14 " .. | " | " | 5 " 21 " |
| 16 | " 22 " .. | " | " | 3 " — |
| 17 | " 24 " .. | " | Negative | — |

A more complete confirmation of the results obtained by the American Commission could not be furnished than the data contained in the foregoing table, since they show that of 17 individuals who were bitten by infected *Stegomyia fasciata*, 8 (47 per cent.) developed the disease. Most unfortunately in three of these cases very grave symptoms ensued, such as black vomit and suppression of the urine, which eventuated in the death of the patients. I may add that in the hands of Guit  ras *fomites* fail to exert any effect on non-immunes.

Whether other species of mosquitoes than *Stegomyia* are capable of conveying the parasite of yellow fever has not as yet been determined by the Commission;

nor have we been able to ascertain whether the parasite passes from the mother insect to daughter insects. The experiments which we have thus far been able to make for the purpose of determining these important points, although negative, have been too few in number to warrant any definite expression of opinion.

The Prevention of Yellow Fever.

The definite determination of the way in which yellow fever is transmitted from the sick to the well, furnishes a solution at last of that much vexed problem of how to prevent the spread of the disease. Even in the absence of more definite knowledge concerning its specific agent—knowledge greatly to be desired from a scientific standpoint—we are now able, as sanitarians, to direct our efforts along certain well-defined lines, with a feeling of security heretofore unknown.

From the point of view of prevention the situation may be briefly summed up in the following conclusion, which was presented by the American Army Commission to the Pan-American Congress of 1900*, viz.: "The spread of yellow fever can be most effectually controlled by measures directed to the destruction of mosquitoes and the protection of the sick against the bites of these insects."

This conclusion was the logical outcome of the observations that had been made by the Commission at its Experimental Station near Quemados, Cuba.

The importance of the discovery that yellow fever is transmitted by the bite of a certain species of mosquito did not fail to attract the prompt attention of the Military Governor of the Island of Cuba, himself a physician and formerly a distinguished member of the Medical Department of the United States Army. By his direction the theory was at once subjected to a practical test in the city of Havana, in which city yellow fever had not failed to make its yearly appearance during the past one hundred and forty years.

Under the efficient management of the Chief Sanitary Officer, Surgeon-Major Wm. C. Gorgas, U.S. Army, the sanitary regulations were so far modified as to require that every patient having yellow fever should not only be quarantined, but that his room should be promptly protected with wire-screens, so as to prevent the possibility of mosquitoes becoming infected by sucking the blood of the patient. As a second important measure, a systematic destruction of all mosquitoes in other rooms of the patient's house, as well as in adjoining houses, was at once begun, the fumes of *pyrethrum* being relied upon to stupefy the insects, after which they were carefully swept up and burned. In other words, Surgeon-Major Gorgas, relying upon the well-known slow progress of yellow fever, sought to destroy all mosquitoes, infected or non-infected, within a given radius of each case, while at the same time he effectually excluded all mosquitoes from access to the sick. If a secondary case occurred, the same hygienic measures were vigorously enforced along the lines above indicated.

As an illustration of what has been accomplished by these newer sanitary regulations, I may state that counting from the date when they were put into force,

viz., February 15th, 1901, Havana was freed from yellow fever within ninety days; so that from May 7th to July 1st—a period of fifty-four days—no cases occurred. Notwithstanding the fact that on the latter date and during the months of July, August, and September, the disease was repeatedly reintroduced into Havana from an inland town, no difficulty was encountered in promptly stamping it out by the same measures of sanitation intelligently applied both in the city of Havana as well as in the town of Santiago de las Vegas, whence the disease was being brought into Havana.

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Obituary.

WE regret to learn that Charles James Hill Wray, the Medical Officer of Health for the town and port of Brisbane, Queensland, Australia, has succumbed to plague. Dr. Wray was educated at Queen's College, Belfast, and held the diplomas of the College of Surgeons and Physicians of Edinburgh, where he qualified in 1870. Brisbane has at the present time a considerable outbreak of plague, entailing a great amount of work on an officer who, like Dr. Wray, held the post of medical officer for both the town and port; not only has accommodation, equipment and attendance, &c., to be provided for the actual sufferers, but the inspection of ships entering the harbour has to be rigorously attended to. Only those who have seen what a plague outbreak means in an important shipping port can gather the work thrown upon a medical officer in Dr. Wray's position. Before further medical assistance can be procured the tax upon the

* *Loc. cit.*

Health Officer at the initiation is enormous, and it is evident Dr. Wray has sacrificed his life in his desire to do all he could to limit the outbreak and stay an epidemic.

Review.

THE CAUSATION AND PREVENTION OF MALARIAL FEVERS. A Statement of the Results of Recent Researches. Drawn up for the use of Assistant Surgeons and Hospital Assistants. By Captain S. P. James, M.B. (Lond.), I.M.S. Illustrated. Pp. 18. Simla, 1902.

Captain James, one of the members of the Royal Society's Commission on Malaria in India, has done a great public service by publishing this excellent pamphlet. It is most important that the class of men for whom the pamphlet is intended should combine intelligence with the more mechanical duties they are called upon to perform. It is impossible for these men, unless they are wisely instructed, to become so; but with Captain James's work in their hands, they cannot fail to gather the meaning and importance of the attempt to arrest malarial infection. The opening sentence is significant: "Malaria is the most important disease of India, and directly and indirectly accounts for more deaths than any other disease." A danger known and faced is already begun to be combated, and Captain James's plain statement declares the power and strength of the enemy. With this pamphlet non-medical men can recognise the different forms of mosquitoes, can gather their habits and become acquainted with their breeding places.

The methods of getting rid of and preventing malarial fevers are referred to under three heads:—

- (1) The extermination of *Anopheles* mosquitoes.
- (2) The administration of quinine.
- (3) Isolation.

Excellent plates of the female *Anopheles*, of the heads of male and female *Anopheles*, of an *Anopheles*' egg, and of a full-grown larva drawn by Dr. S. R. Christophers, together with the wings of mosquitoes, form a useful addendum to a most welcome and important publication.

We hope the Indian Government authorities will see that this pamphlet is allowed to be sold in all the British Possessions where malaria prevails.

Current Literature.

THE SURGICAL PECULIARITIES OF THE AMERICAN NEGRO. By Rodolph Matas, M.D., Louisiana.

After a statistical inquiry based on the records of the Charity Hospital of New Orleans, U.S.A., decennium 1884—1894, Dr. Matas comes to the following conclusions:—

(1) The North American negro, as he is known at present in the United States, is anthropologically, physiologically, and pathologically different from his original African ancestors and from his uncivilised

brothers in the West Coast of Africa of the present generation.

(2) A residence of nearly three hundred years on the Southern States of North America in contact with the white man and under the influence of civilisation has produced a marked change in the mental and physical organisation of the negro.

(3) This change is evidently due to the combined influences of acclimatisation and adaptation to surroundings other than those of climate, and especially to miscegenation with the white race.

(4) That the general morbidity and mortality of the coloured race was less than that of the white population in the South during the whole period of slavery and up to emancipation.

(5) That since the coloured race has been thrown upon its own resources (since 1864) its morbidity and mortality have enormously increased, and are now much greater than those of the white population.

(6) That in consequence of the altered conditions of existence the diseases which were peculiar to the slave period, and notably the more typical African diseases, are rapidly disappearing. On the other hand, the general liability of the negro to the common diseases of this country is rapidly increasing. So that many immunities which he formerly enjoyed have been lost and new predispositions to disease have been acquired. In other words, the tendency of the coloured race is to lose the specific pathological peculiarities which it acquired during the original process of race differentiation in Africa, and to rapidly subject itself to the conditions that affect the white race.

(7) There are no diseases which prevail *exclusively* in the coloured race any more than there are diseases which prevail *exclusively* in the white race. The differences, pathologically speaking, that do exist between the white and the coloured population lie only in the *relative* predisposition to some of the diseases that prevail in this country and in their *relative* immunity from others.

(8) When viewed from the purely surgical operative standpoint, the white and the negro are practically alike, especially when individuals of both races, taken from the same social environment, are compared. There are no apparent differences between the races on the operating table. The same technique applies to both equally well; and often, especially in the matter of resistance to shock, the negro appears to better advantage than the white man. In the general and local reactions of the tissues to infection there are some differences between the races. It is in the histogenetic tendencies of the tissues that we find the real surgical contrast between them. If we are to judge from this alone, the coloured race reveals in this last particular a marked tendency to degeneration.

(9) That the progress accomplished in modern surgery by the introduction of anaesthesia and antiseptics have proved just as applicable and advantageous to the negro as to the white man.

(10) That the comparative statistics furnished by the records of the Charity Hospital of New Orleans for the last decennium on the surgical diseases of the coloured race confirm, in general, the conclusions of the United States Census of 1890, and are in harmony with the general impression that prevails throughout

the South, viz., that the coloured race is degenerating, if by this we mean a growing inability to resist the causes that are inimical to its existence.

(11) That the degenerative tendencies of the coloured race revealed by statistics, are due, essentially, to the influence of unfavourable hygienic surroundings; to unfavourable social (including moral) environment; to all the causes which lead to a bad heredity, vice, dependency, and degradation, and which are acting simultaneously upon an ethnologically inferior and passive race which is struggling for existence with a superior, aggressive, and dominant population.

MALARIA.

MALARIA AND MOSQUITOES.—The Japanese papers publish the results of experiments made by their military authorities to confirm the mode of infection of malaria. One battalion of soldiers in Formosa were efficiently protected for five months from the bites of mosquitoes during the entire malarial season, with the convincing result that the soldiers remained perfectly healthy. In another battalion who were not subjected to any protection or precautions and who were quartered in the same place and for the same time, 259 cases of malaria occurred. This experiment is not alone interesting from the point of view of the malaria-mosquito theory, in establishing in a striking manner that the mosquito is wholly and solely responsible for malaria, but it demonstrates how advanced the Japanese are in their study of medical science, and how ready to put the knowledge they have acquired to a practical test.

LATENT MALARIA.—George B. Young, M.D., in the *Amer. Pract. and News*, March 15th, 1902, divides latent malaria into three groups: (1) Cases of long incubation before the appearance of fever; (2) cases in which no pronounced clinical manifestations are manifested; (3) cases in which the determining cause of the onset of the fever is due to some cause irrespective of malarial infection.

There are several theories extant as to the cause of delay or suppression in the manifestations of malarial infection. (a) That the parasites lie dormant in the spleen, or bone, or marrow; (b) that they multiply so slowly that they fail to produce a febrile reaction; (c) that an entirely different cycle may be followed by malarial parasites under certain conditions. Under these circumstances it is incumbent to examine the blood for malarial parasites in almost all the less pronounced ailments met with in the malarial districts.

TYPHOID FEVER.

THE TYPHOID BACILLUS.—In an address published on typhoid fever, by Victor C. Vaughan, M.D., in the *Journal of the American Medical Association*, April 19th, 1902, the following conclusions, after a systematic study of all toxicogenic germs found in samples of drinking water, suspected of causing typhoid fever, are announced:—

(1) A water containing a typical colon bacillus, one

which coagulates milk within from twenty-four to forty-eight hours, readily reddens litmus and produces an abundance of gas in glucose media, does not cause typhoid fever. There are several smaller cities in Michigan which send to us monthly their drinking water supply for bacteriological examination, and some of these waters contain the colon bacillus constantly; in fact, this germ never fails to appear when a test is made, and yet there have been no epidemics of typhoid fever among the people drinking these waters.

(2) The more markedly a germ found in drinking water differs from the typical colon bacillus, and the more closely it approaches the typical typhoid germ, the more likely is typhoid fever to appear among those using the water containing this germ. I am not ready, even after fourteen years of investigation of this matter, to lay down any rules by means of which I can say just where the line should be drawn between the colon and the typhoid groups, and I still continue to speak of typhoid-like bacilli, and I have no hesitancy in condemning waters which contain these micro-organisms.

(3) I have never found in any sample of drinking water a typical Eberth bacillus. I do not say that such a germ may not be found in water, but I do not believe that the typhoid bacillus preserves its typical characteristics for any great length of time when growing in water. As confirmatory to this last statement, it may be worth while to mention the fact that Remy has found that when the typhoid bacillus is grown in company with the colon germ the former is so far changed that it cannot be identified by any means at our command at present, and that it even loses its susceptibility to the agglutinating action of typhoid serum.

SCABIES (ITCH).—Kaposi's ointment for scabies:—

| | | | | | | |
|-------------------|-------------------------------|-----|-----|-----|-----|-------|
| R. Beta-naphthol | ... | ... | ... | ... | ... | 5iii |
| Cretæ prep. | ... | ... | ... | ... | ... | 5iiss |
| Saponis mollis | ... | ... | ... | ... | ... | 5iii |
| Adipis | ... | ... | ... | ... | ... | 5vi |
| M. Ft. unguentum. | Sig.: Apply locally at night. | | | | | |

TREATMENT OF BLACKWATER FEVER.—Dr. R. O'Sullivan-Beure states that in equatorial East Africa a decoction of "cussia abbreviata" is much extolled in the treatment of blackwater fever. This observer regards blackwater fever as a disease apart from malaria, although the two diseases may be combined in the same person.

CHOLERA.—The Philippines.—Up to April 20th, 1,244 cases of cholera were reported in the Philippines, of which number 902 died. Of this number 388 cases occurred in the city of Manila, with 300 deaths.

PLAGUE.

PREVALENCE OF THE DISEASE.

INDIA.—The deaths from plague recorded in India during the weeks ending April 5th, 12th and 19th, amounted to 23,286, 24,380, and 19,412 respectively. By telegram we learn that in Bombay city during the month of April of this year 2,833 persons died of

plague. In the districts of the Bombay Presidency, Kaira, with 1,061 deaths; Khandesh, with 1,179 deaths; Satara, with 981; Belgaum, with 720; Kathiawar with 1,823, and Cutch, with 572 deaths from plague during April, were the localities in which the disease chiefly prevailed.

The Punjab is still by far the most seriously affected district of India. During the weeks ending April 5th, 12th and 19th, the number of deaths from plague in the Punjab amounted to 14,871, 16,281, and 14,145 respectively. In other parts of India the deaths from plague during the weeks ending April 5th, 12th and 19th, numbered respectively: Calcutta, 689, 603, and 588; Bengal, 788, 603, and 760; North-West Provinces and Oudh, 1,570, 1,660 and 773. In other parts of India the disease is less prevalent, and in many instances, especially in the South, the advance of the summer seems to coincide with a gradual lessening in virulence of plague.

EGYPT.—During the week ending April 20th, 27th, and May 4th, the fresh cases of plague in Egypt numbered 31, 18, and 22, and the deaths from plague during the same period, were returned as 19, 8, and 15 respectively.

CAPE OF GOOD HOPE.—Port Elizabeth is the only town or district of Cape Colony in which plague cases have occurred for two months past. The numbers are, however, quite insignificant. During the weeks ending April 5th and 12th, the number of fresh cases amounted to 2 and 5 respectively, and the deaths from the disease to 2 and 3.

HONG KONG.—During the weeks ending April 19th, 26th, May 3rd and 10th, the number of fresh cases of plague in Hong Kong numbered 7, 5, 28, and 24; the deaths from the disease during the same periods amounted to 6, 5, 26, and 19 respectively.

AUSTRALIA.—A telegram dated Sydney, May 14th, states that there have been 126 cases of plague in Sydney, and 35 deaths from the disease since the present recurrence commenced. On May 14th forty cases of plague were in Hospital. By a telegram dated May 8th, we learn that 35 cases of plague were in Hospital in Brisbane, Queensland.

ARGENTINE.—A few cases of plague are reported in the Argentine, but the outbreak is quite limited.

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Médecine Navale.
Archives Russes de Pathologie, de Médec. Clinique et de Bactériologie.
Australasian Medical Gazette.
Boletín de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Brooklyn Medical Journal.
Caducée.

Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Esercito
Hong Kong Telegraph.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
Journal of the American Medical Association.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Medical Record.
Medical Review.
Merck's Archives.
New York Medical Journal.
New York Post-Graduate.
Pacific Medical Journal.
Polyclinic.
Public Health.
Revista de Medicina Tropical.
Revista Medica de S. Paulo.
Sei-i-Kwai Medical Journal.
The Hospital.
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The Journal of Tropical Medicine.

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Original Communications.

THREE LECTURES ON BILHARZIA,

Delivered at Kasr-el-Ainy Hospital, Cairo.

By FRANK MILTON, M.R.C.S.

Surgeon to the Hospital.

LECTURE I.

GENTLEMEN,—The disease which amongst us is generally spoken of as bilharzia and whose chief symptoms are manifested through changes in the bladder and large intestine, is due to the deposition in the tissues of the eggs of a trematoid worm, the *Bilharzia hæmatobia*, whose favourite habitat is the portal vein.

THE WORMS AND THEIR OVA.

Both *male and female worms* are found, the size and appearance of the two sexes being very different, the male being short and thick, measuring about 15 millimetres in length and rather more than 1 millimetre in breadth, the female is long and filiform, being about 20 millimetres long and not nearly so thick as the male. Although the portal vein is the part in which the worms are most commonly found and the place where they are looked for in the routine performance of *post-mortem* examination, yet it must never be forgotten that they are common also in the hæmorrhoidal and vesical veins, and have been found in widely separated parts of the body and may probably inhabit any part of the venous system. The whole of the various manifestations of the disease known under the name of bilharziosis are, as we have said, due primarily to the deposition of the eggs of these worms in the tissues of the part; these eggs are translucent oval bodies, about 0.16 millimetre in length and 0.06 millimetre in breadth; they are smooth surfaced and are readily recognised by their possessing a spine situated as a rule at one end, but which under certain conditions may be placed laterally, this latter occurring

frequently in eggs deposited in the rectum, whereas, the eggs recovered from the bladder invariably have their spine at the end. This variation in the situation of the spine has not yet been satisfactorily accounted for any more than has the function of the spine itself been decided; but whatever may be the cause of its various position, I think its function is probably to aid the egg in its task of escaping from the blood-vessels and penetrating the surrounding tissues until it makes its way to a surface from which it can be discharged from the body to continue its cycle of changes until it returns to its human host to be finally developed into the mature worm. What these changes are that occur after the egg is discharged from the human body is not yet clearly known, nor has it been discovered with certainty how the embryos of the worms gain readmission to the body.

MODE OF INFECTION.

Many quaint ways have been suggested as to how the embryos enter the body; some assert that the worms themselves enter by the anus or urethra of the victim during bathing, or even by the eggs making their way through unbroken skin through washing in infected water. All ideas of infection clearly point to water as being the medium through which the infected matter is conveyed, and it is almost certain that water is the vehicle, and that infection is brought about by drinking and by no other means. Segmentation of the yolk is said to begin in the uterus of the female bilharzia, and the eggs when discharged in the urine often contain a well-formed embryo, and even embryos have been found which have hatched out in the bladder, but when this has taken place the embryos have invariably died after a very short period, and have been of no importance from a pathological point of view, nor as helping to indicate their later life history.

By far the commonest seat of "*bilharzia*" is the urinary bladder, and it will be most convenient if we

consider the disease in connection with this viscus in some detail and then enumerate more briefly the changes which take place in the less commonly affected tissues. As a beginning of the disease we must, of course, have had the bilharzia worms introduced into the body of the host where they have established themselves and set to work to produce their eggs. The period of life and sexual activity of the bilharzia worm is not known, but it is probably of considerable duration, for cases have been recorded of patients who have ceased to inhabit countries where bilharzia is known to exist, continuing to pass living eggs after years of residence under circumstances where reinfection would appear to be an impossibility. Sonsino has recorded the case of an Egyptian student in France who, after nine years' uninterrupted residence in that country, was still passing living eggs in his urine. The eggs when laid by the female bilharzia are free in the blood-vessels and drift until they are arrested in some capillary whose calibre is too small to let them pass; this would seem to imply drifting against the current of the blood, as the eggs are produced in the veins where of course the current of blood is from small to larger. But I may perhaps be allowed to speak of drifting in the absence of any certain knowledge of how the eggs get from their point of origin in the larger veins to the smallest capillaries; that they do get there is certain, and that they do not make the entire circuit of systemic circulation is also certain, and we must be satisfied with these two facts.

MODE OF EXIT.

As the life of the embryo has to be passed outside the body of the host of the parent worm, Nature directs the eggs towards the nearest point of exit from the body, and this will be the nearest surface from which they may be discharged; thus it will be found that the eggs are directed towards the mucous membrane of the bladder, in the capillaries of which they become impacted.

CHANGES PRODUCED IN THE BLADDER.

Many eggs out of the enormous number produced go astray, and instead of finding their way into the mucous and submucous coats become lodged in the muscular, and even serous, coats of the viscus, where they give rise to a peculiar train of symptoms which will be referred to later on. At present we need only concern ourselves with the changes which take place in the internal coats of the bladder. At first, then, we have isolated eggs deposited here and there in the mucous and submucous coats; whilst these are comparatively few in number their presence would be difficult to detect, and they give rise to no symptoms; but as the parent worms go on industriously laying their eggs and discharging them into the bloodstream, more and more of these eggs find their way into the capillaries which are already obstructed by the earlier arrivals, and areas are formed in which the eggs are found closely packed together, and not only plugging the minute vessels, but many will have made their way through the coats of these latter and lie in the surrounding tissue. These deposits of eggs, when they have increased in size sufficiently to be

visible to the naked eye, will appear as small pellucid bodies situated in the mucous membrane and raising its surface above the surrounding level. As more eggs are deposited these patches increase in size, and neighbouring ones coalesce to form larger patches, until eventually the whole mucous membrane of the bladder may become involved. The most frequently and the earliest affected part is the base of the bladder, especially the trigone, and from here the affection spreads outwards. As the patches increase in size they also increase in thickness, owing to the overgrowth of the surrounding tissue due to the irritation of the presence of the eggs and also to the fact that Nature, striving to undo the mischief she has permitted for the sake of the propagation of the bilharzia species, tries to render the eggs harmless within the host by enveloping them in fibrous tissue and isolating them, as she does to foreign bodies embedded in living tissue elsewhere. These two processes, the irritative overgrowth of the normal tissue and the formation of new fibrous tissue, give rise to the formation of extensive raised patches of a yellow-grey or brownish tinge with a granulated surface and of hard consistence, whose feel, when explored by a sound, so soon becomes familiar to you in the outpatient room, and which is always pathognomonic of bilharzia. These patches, when they have attained a certain age, begin to lose their vitality, partly by the mechanical interference with their circulation and partly by the contraction of the new fibrous tissue of which they are largely composed, and as a result they begin to break down and even to slough, giving rise to ulcers and crevices on their surface which tend to retain a certain amount of urine in their cavities, and to set up decomposition in it and deposition of its salts. It would appear that in many cases the increase in the mucous membrane due to irritative hyperplasia is out of proportion to the development of fibrous tissue, and in these cases occur great overgrowth of the normal structure of the mucous membrane with formation of villousities, polypoid growths of great vascularity, and more solid cockscomb-like tumours, which are all equally typical of the disease. These growths, although they are very plentifully supplied with blood-vessels, are all liable to necrosis and ulceration from blocking of their capillaries by the bilharzia eggs with which they swarm, and their disintegration, as well as that of the thickened patches, gives rise to the typical bilharzia urine containing blood, pus, minute sloughs and bilharzia eggs entangled in mucus. It has been stated that mature worms have been found in dilatations of the veins in these polypoid growths, which would account for the enormous quantities of eggs and great overgrowth of tissue which occur in these cases.

CARCINOMA AND BILHARZIA.

Besides the fibrous growth occurring in bad bilharzia cases, true carcinoma is also sometimes found grafted on to the existing disease probably beginning in an irritative overgrowth of the glandular elements of the mucous membrane; this co-existence of carcinoma and bilharzia is generally regarded as fairly common, but I think it does not in reality occur so frequently as some writers would lead one to expect,

and what has been regarded as tumour due to cancer is, I think, in many cases, simply tumour due to excessive formation of what may be termed bilharzial tissue. This bilharzial growth, to which we shall so often have to refer, is in the later stages of the disease developed to a most extraordinary degree. It originates, of course, in the bilharzia patches described before, but the dense, thick masses of new tissue into which they develop lose all resemblance to the excrescences first formed. Unfortunately I have never yet been able to trace the development from the one form into the other continuously, but it would seem as if the new tissue after a time was formed too quickly to be properly organised, and the result is the formation of this dense, structureless mass which breaks down before the finger introduced into the bladder with resistance equal to recent blood-clot into approximately cubical rectilinear-sided masses having the appearance of pinkish cream cheese. Its amount is often very large, as it may form the sole contents of the distended bladder. Its vitality must be very low, and its destruction causes but little hæmorrhage. These excessive formations of new tissue in the mucous membrane of the bladder naturally interfere with its functions as a contractile viscus, and not only is it rendered incapable of properly expelling its contents, but there is also obstruction to the entrance of urine from the ureter; this in its turn causes dilatation of the ureters, which in process of time extend through their whole length and reacts on the pelvis of the kidneys, which become dilated and causes gradual disorganisation of the kidneys themselves. When it happens, as is so frequently the case, that the contents of the bladder are represented by a collection of decomposing organic tissue and stinking urine, the step from simple dilatation of ureters and hydronephrosis to septic pyonephrosis is very easily taken.

THE URETERS AND KIDNEYS.

The ureters and kidneys may themselves be directly affected by bilharzia, but this does not happen nearly so frequently as primary affection of the bladder, and it would appear as if the further the part was removed from the bladder the less likely was it to be attacked by the disease. Thus the ureters are fairly commonly affected in their lower third, the middle and upper thirds are rarely affected, the pelvis of the kidney more rarely, and the tissue of the kidney itself more rarely still; but cases have been described where eggs have been found deposited in its secreting structure.

At the other end of the genito-urinary tract the seminal vesicles are said to be frequently and severely affected, but for some reason or other we do not seem to have our attention drawn to this form of bilharzial affection at this hospital, nor do I ever remember to have met with it in private practice, and I think we need not give very much attention to its pathology.

Proceeding forward from the bladder we come to a part which is frequently and severely affected, and which is of the greatest interest to us as practical surgeons, for in it is found the form of disease which, though as a rule not directly threatening life, entails a vast amount of suffering in its victims, and which

is the form of the disease which is most amenable to surgical treatment—and that part is the urethra.

URETHRAL FISTULA.

The ultimate manifestation of bilharzia in the urethra takes the form of urethral urinary fistula, and it is on account of urinary fistula that so many cases of bilharzia come for treatment. It is interesting to note in this connection that of all the many cases of urinary fistula treated in my wards in Kasr-el-Ainy, and of all the cases coming for treatment in the out-patient department—and these must be counted by thousands rather than by hundreds, the invariable cause of fistula has been bilharzia. Even the few cases of perineal fistula after operation for stone, which have come from time to time, have had bilharzia as the primary cause of their malady, and I think I may safely say that I have never seen a case of urethral urinary fistula in a native of this country that did not owe its origin to bilharzia.

The number of cases of urinary fistula presenting themselves for treatment is very great, so much so that at one time it seemed as if the general usefulness of the surgical side of the hospital would be seriously impaired through the cases of fistula usurping all the available beds. It was therefore necessary to restrict the number of cases under treatment at one time to ten beds, or about 10 per cent. of the total male surgical accommodation. Those who have to dress the cases very well know these beds are invariably full, and only the resident assistant surgeon knows how many cases there are constantly waiting for admission in order to be relieved of this truly horrible disease, which renders its victims a nuisance and an offence not only to their neighbours but also to themselves, from the loathsome state of filth and stench which is unbearable even in this land of many odours.

The favourite situation for these urinary fistulæ is in the perineum, near the scrotum and on either side of the middle line, where the vast majority of fistulæ will be found. The next most favourite place is in the posterior part of the scrotum itself, but the fistulæ here are generally multiple, and more often than not accompanied by fistulæ in the perineum. Apart from these situations the fistulæ may occur in the anterior part of the scrotum, or in the penis immediately above the scrotum—but these are not common, and anteriorly to this point in the penis itself, although they do occur, they are excessively rare. Away from the course of the urethra itself they are found near the anus, in the ischio-rectal fossæ, above the pubes, and in the upper third of the adductor region of the thighs, but this only in very severe cases with many fistulæ. Although the external openings of the fistulæ may be distributed over a very wide area of the skin surface, practically the whole of them are limited in their origin to the part of the urethra immediately in front of the bulb. It will be found also that by far the greater number of these fistulæ take their origin from the pubic side or roof of the urethra; a small number will be found to take their origin from the perineal side or floor of the canal, but these are quite the exception, being probably in the proportion of less than one to twenty of the roof fistulæ.

It would seem, at first sight, a needless splitting of straws to distinguish between fistulæ arising from the roof of the urethra and fistulæ arising from the floor, but the whole history, manner of formation, signs, symptoms, and sequelæ are different for the two forms, and more than warrant a practical distinction and description apart.

The *floor fistulæ*, although the majority are formed in the part of the urethra just in front of the bulb, are by no means so strictly confined in their range as are the roof fistulæ, and they may be found fairly frequently in the penile urethra. It would seem natural enough that the neighbourhood of the bulb should be chosen as the part of the urethra most frequently to be attacked by bilharzial disease, as in it is the largest collection of venous plexuses offering the most commodious resting-places for the parent worms, but it is not so clear why the roof of the urethra should be so persistently chosen for the point of attack rather than the floor. It is worthy of note here that although hundreds of cases come to hospital every year with presumably bilharzia worms in their corpora spongiosa, as indicated by urethral fistula, no one, as far as I know, has ever presented himself with evidence of the neighbouring venous plexuses of the corpora cavernosa being affected, and this is the more remarkable when we recollect the near connection between the penile and vesical plexuses of veins.

But to return to our subject. The method of formation of these urethral fistulæ is as follows: The first stage which is common to the two forms of the disease exactly resembles the process of formation of a patch of infiltration and infarction in the bladder; that is to say, the eggs are deposited in the submucous and mucous coats, and give rise to proliferation and overgrowth of these tissues, with later on death of small areas of the invaded part, due to blocking of the capillaries and to pressure from contraction of new fibrous tissue. Up to this point the process is common to all forms of bilharzia invasion, but after this point the disease follows a special course and varies according as the part affected is the roof or the floor of the urethra. In the *roof of the urethra* the infarction, as the mucous and submucous coats are destroyed, extends more deeply into the substance of the corpus spongiosum itself, and as the infarction proceeds so does the destruction of tissue in its wake, until a track has been destroyed down to the limiting membrane of the corpus spongiosum. This, in its turn, is attacked, and the disease breaks through into the space between it and the corpora cavernosa. Here the advance would appear to halt for a while, for in this position there is always found an accumulation of the peculiar white unhealthy granulation tissue which is formed in the track of these fistulæ. The disease soon advances again, but at this point it takes a new direction, turning round the outside of the corpus spongiosum and making towards the skin surface in the perineum where, having arrived, the skin is destroyed in its turn, an opening formed, and the fistula is complete.

It is not very easy to see at first why the infarction and destruction of tissue in urethral bilharzia should extend deeply into the underlying parts instead of following the ordinary course of the disease elsewhere

and extend along the surface, but the probability is that the fluid contents of the urethra, being in motion and under pressure, tend to break down the weakened tissue in the walls of the canal and to open up fresh spaces for depositing wandering eggs and detritus. The amount of inflammation involved in this process of formation of a roof fistula is very small, and the symptoms it gives rise to are not pronounced, but in the formation of a floor fistula the case is very different. Here, for some reason or another, the infarction and consequent destruction of tissue occurs in the perineal surface or floor of the urethra with the formation of an ulcer; the ulcer being in a dependent position would appear to act as a trap, catching and retaining dead, and it may be decomposing, matter, either from the bladder or from its own neighbourhood. These matters, as they collect and as the ulcer extends in depth, set up a septic inflammation with formation of pus and infection and decomposition of any drops of urine which have infiltrated the part and been detained. In this way is speedily formed a peri-urethral abscess, continually receiving fresh supplies of decomposable material from the proximal urethra, and as continuously pouring out an extremely irritating mixture of septic pus and decomposed urine into the distal urethra. This state of septic inflammation with retention in the cavity of the abscess of decomposed and irritating matter, but without tension, owing to the continual escape of pus into the urethra, causes a great proliferation of the fibrous tissue elements in the immediate neighbourhood of the abscess until a fibrous tumour of stony hardness, and having in its centre a cavity containing stinking pus and *débris*, is formed in direct connection with the urethra. This form of fistula develops much more slowly than the roof variety, and in many cases the pus does not reach the skin surface until the process has been going on for a very long time, and obstruction to the escape of urine along the urethra on the distal side of the fistula has been brought about by secondary change induced in the canal by the septic discharges continuously passing along it.

STRICTURE OF URETHRA.

Indeed, many of these cases come for treatment for the difficulty and pain in micturating and it is only on examination that the characteristic tumour is found. In this connection it must be especially noted that with roof fistulæ stricture is hardly ever found unless the disease is of very long standing and stricture has occurred from actual destruction of part of the urethra and surrounding tissue, caused probably by the breaking down of multiple foci of infarction which is by no means common. With floor fistulæ, however, stricture is invariably the rule, not only in the neighbourhood of the fistula but generally there is marked thickening and narrowing of the urethra in its whole length, from the opening of the fistula to the meatus. Not uncommonly, there is actual obliteration of the canal by cicatricial tissues due to the final healing of long-continued ulceration of the canal set up and maintained by the discharges from the fistula abscess. In cases of roof fistula there may be some difficulty in introducing a catheter into the bladder, but this is due to distortion of the urethra obstructing the passage, and

even in these cases if a full-sized catheter is used in the first instance the obstruction will hardly be noticed.

BILHARZIA IN THE VAGINA.

Before leaving the genito-urinary system it is necessary to refer to the vagina, in which bilharzial affection sometimes occurs. Bilharzia, for some reason not yet understood, is very much rarer in women and girls than in men and boys; only a few cases of bilharzial cystitis in women occur in the practice of the hospital; urethral fistula is practically unknown; and I do not think I have ever seen an advanced case of bilharzia of the rectum in a female. I have, however, seen three or four cases of bilharzial affection of the vagina, so that taking the rarity of the disease as a whole among women, vaginal bilharzia would appear to be one of the commoner forms in the female. The mode of infarction in the vaginal mucous membrane is the same as in the other organs already described, but the subsequent course of the disease would seem to vary. Unfortunately the examples of the disease that I have seen have never been fresh cases, that is to say, they have all been cases that have been for a more or less prolonged period under active treatment and afterwards sent on to me. In all these cases there has been great thickening of the mucous membrane, especially on the posterior surface of the vagina, where the membrane appeared as rugged and heaped up with deep transverse and shallower longitudinal fissures mapping it out into more or less regular areas of thickened tissue. There were no ulcers and no signs of ulcers having existed, but the whole surface was peculiarly dry and harsh, and as if it had been sodden for a long time and then thoroughly dried. This may have been due to some extent to treatment, but I think it was in part, at any rate, characteristic of the disease. In none of the cases that I have seen did the affection extend into the cervical canal, but after all the number of cases observed is but small and not sufficient to draw conclusions from.

INFECTION OF THE RECTUM.

Having referred to all the lesions of the genito-urinary system which are of importance to us as surgeons, we may now turn to the digestive system, which is represented by the rectum where the disease is second only to the bladder in frequency of occurrence, and almost more distressing in its symptoms; and where, although it is less directly threatening to life, the treatment, at present at least, is equally hopeless. As might be expected, the lower end of the intestinal tract, surrounded by a large plexus of veins, and affording all the conditions which would appear most desirable to the adult bilharzial worm, is very commonly the seat of his activities.

The mode of infection and the early stages of the disease affecting the rectum need not be particularly referred to, as they are essentially the same as have been already described in connection with the affection of the bladder, namely, deposition of the eggs in the mucous membrane and submucosa, with infarction of the smaller capillaries and development of new fibrous tissue, overgrowth of the tissues of the part, and localised death of minute portions of tissue.

Although all these changes take place in the rectum, the changes which are most marked and most fully developed are those which make for an increase of growth rather than the changes that bring about necrosis; and thus the disease develops on rather different lines to those followed in the bladder where necrosis is more marked, and the overgrowth of glandular structure is less. Thus in the well established disease the hard indurated patches common in the bladder are never found in the rectum; but instead the mucous membrane becomes hypertrophied and excessively vascular, the surface layers are deeply injected and readily bleed when handled, and the appearance of the surface resembles rich red velvet. After a time the elements of the mucous membrane, probably owing in part to their excessive nutrition, take on abnormal growth with thickening of the structure and general increase in bulk, and with excessive local overgrowths which take the form of polypoid adenomatous tumours. Near the anus these polypoid tumours bear some resemblance to ordinary hæmorrhoids, but within the gut, and especially beyond the internal sphincter, they differ materially from these latter. In the first place in a given area they are infinitely more numerous than piles ever are, and instead of being rounded and smooth like hæmorrhoids their surface is velvety from thickening of the mucous membrane itself, and their outline is broken up in all directions by the formation of secondary polypi growing from their own surface and branching in all directions, until the larger and fully-developed tumours bear a great resemblance to red branching coral. These tumours extend high up the rectum beyond the reach of the finger and this is so invariably the case that it would almost suggest the idea that the affection of the rectum begins from above, in the neighbourhood of the sigmoid flexure, and proceeds downwards; a possibility which has a most important bearing on treatment, as will be seen later on. The infection of other parts of the digestive system, such as the liver, and the consequent production of sclerosis of that organ does not concern us as surgeons, although there have been suggestions of the possibility of a connection between bilharzial infarction of the liver and hepatic abscess; but up till now the connection has never presented itself forcibly to me and I think that for the present we may allow the question to stand over. Bilharzial infarctions giving rise to abscesses and sinuses occur in what might be called irregular positions. Thus in a very interesting case of multiple superficial sinuses in the sacral region, of a boy, aged 12, for which no cause was very apparent, I was so struck by the peculiar appearance of the granulation tissue removed when they were scraped out, that I sent a specimen to Dr. Symmers, who reported on it as being full of bilharzia eggs, and this although the boy had no signs of bilharzia either in his rectum or bladder.

Dr. Symmers also once showed me a tumour the size of half a walnut, which he had removed *post mortem* from the free edge of the broad ligament of a young girl, and which was made up entirely of bilharzia eggs and young fibrous tissue. These cases are to be especially noted as re-enforcing the fact which is always in danger of being forgotten, that bilharzial

infection is not confined to the urinary and digestive systems, but may occur in any tissue or part of the body.

Gentlemen, having to-day concluded my remarks on the pathology of this most interesting disease, on the next occasion we will discuss its symptomatology and diagnosis.

THREE CASES OF TUMOUR OF THE NOSE.

By FRANK COLE MADDEN, M.B., B.S.(Melb.), F.R.C.S.

Professor of Surgery Egyptian Government School of Medicine, and Junior Surgeon Kasr-el-Ainy Hospital, Cairo.

IN the issue of the JOURNAL OF TROPICAL MEDICINE for May 1st, 1902, I find a series of photographs of a peculiar disease of the nose, which resembles some cases of a somewhat similar nature which I had at Kasr-el-Ainy Hospital last year, and I venture to forward you photographs of them for publication.

Photograph I. represents an Egyptian woman, aged 35, who came for treatment originally for a small, hard, mucous-like polypus, which was projecting from the anterior nares. The nose gradually expanded, became flattened, and presented a peculiar saddle-shaped appearance, the skin at the same time becoming hard and thick, very like pig-skin, with much hypertrophied sebaceous glands, and a wide separation between the individual hair follicles. The opening of the nostrils soon became completely blocked, and a thick, hard collar of fleshy substance protruded. The upper lip was never at any time affected, but there was a small hole in the hard palate, but without any ulceration. The condition gradually developed and took nearly two years to reach the condition illustrated in the photograph. The patient would neither enter hospital or submit to any operation, and I have now lost sight of her. The interesting point, however, is that the other two cases seemed to start in very much the same way, but progressed much more rapidly.

Photograph II. is that of a Soudanese man, who was admitted to hospital on April 11th, 1901, in the condition depicted. He stated that it began as a small nodule inside the right nostril, which gradually increased in size. He had suffered much at the hands of many native "barbers," with cauteries, scrapings, scissors, &c., before he was admitted, and all to no purpose. As will be seen, the disease had broken down and had affected the whole of the anterior part of the nose, as an ulcerated, protruberant mass, with thickened, infiltrated skin at the margins. The front part of the septum is also involved, and there is an ulcerated surface covered with granulations of an unhealthy character in the mouth between the upper lip and the gums over the central incisors. The unaffected portion of the nose is hard, and the skin is in exactly the same condition as Case I., with the same widening, flattening, and pig-skin appearance. A small piece of the growth from the gums was examined and found to be a round-celled sarcoma.

The whole of the mass was removed, the free margin of the lip being left. The result of the opera-

tion was very good, though a future operation for a new nose will be necessary. There was no sign of recurrence several months after the operation.

Photographs III. and IV. represent a far more severe case of the same condition. She was an Egyptian woman, aged 32, who dated her illness from a fall on the nose; but, on closer examination, she stated that the small fibrous tumour, seen to the right and upper side of the mass, came first, and I have no doubt the disease started in much the same way as the others. The general appearances are just the same, but there was an ulcerating mass, involving the greater part of the hard palate and filling up the space between the alveolar processes of the upper jaw. The red margin of the lip was not affected, as is seen in photograph IV.

After performing preliminary tracheotomy, I was able to remove the whole mass in its entirety, though it ran back as far as the naso-pharynx and involved the whole of the hard palate. I subsequently tried to make a new nose, but with only partial success.

These three cases resembled rhinoscleroma, but there was no affection of the upper lip, and no special tendency to spread in the substance of the skin or mucous membrane. Actinomycosis was also suggested, but examination of the pus gave a negative result. Syphilis, lupus, and tubercle were also possibilities, but the cases were pronounced to be sarcomatous by the examination of a piece of the tumour. The last two cases also illustrate the difficulties under which "tropical" surgeons work, for it is hardly possible to realise that any English patient would allow his disease to assume such proportions before seeking medical advice.

SLEEPING SICKNESS; A FORM OF CEREBRAL ELEPHANTIASIS.

A SUGGESTION BY OSBORNE BROWN, M.B.

Assist. Col. Surgeon, Elmina Castle, Gold Coast.

I WOULD like to make a suggestion regarding the etiology of sleeping sickness. It is only a suggestion, and I make it in order that those who may come across cases of this disease may follow it up.

In some cases the embryonic form of *Filaria perstans* was found in the blood. May the disease not be caused by the parental form of this or other embryos circulating in the blood occluding the lymphatics leading from the brain, causing chronic inflammatory changes there similar to elephantiasis in any other part of the body? In other words, may not sleeping sickness be a sort of cerebral elephantiasis? We know that there is perivascular inflammation and degeneration of nervous elements, with appearance of cholin in the circulation.

DR. D. C. REES has been appointed Medical Inspector on the Staff of the Medical Officer of Health for Cape Colony. It will be remembered that Dr. Rees formerly held the appointment of Medical Superintendent of the London School of Tropical Medicine.

THE SLEEPING DISEASE (DOENÇA DA SOMNO).

From the Portuguese.

REPORT SENT TO THE PORTUGUESE MINISTER OF MARINE BY THE SCIENTIFIC COMMITTEE SENT TO STUDY THE SLEEPING SICKNESS IN WEST AFRICA, ON FEBRUARY 21ST, 1901.

(Continued from page 151.)

PART II. (condensed).

We have up to the present observed clinically 28 individuals attacked by the sleeping disease, 22 males and 6 females, including the 5 cases we studied during our residence in the Island of the Principe. Of the 28 cases, 13 were adults, and 15 between 7 and 11 years.

The patients under observation in Loanda were 11 natives of Donda, 3 of Libollo, 3 of Quissama districts near to the River Quanza, 1 from Caboverde, 1 from Malange, residing in Dordo, 1 from Gelungo Alto, 1 from Beneguella, 1 from Sierra Leone, residing in Loanda, 1 from this city; the others lived in Josudo, near the river mouth. Of all the patients 12 died, and all were examined *post mortem*.

DURATION OF THE DISEASE.

Judging from the few cases we were able to gain reliable information concerning, the duration of the disease seems to be about three to four months.

SIGNS AND SYMPTOMS.

Sleeping sickness shows itself principally in notable perturbations of the lymphatic, the nervous, the circulatory, and the calorific systems.

There are always **glandular swellings**, sometimes limited to the region of the neck, but sometimes occurring as a general swelling, invading more or less the lymphatic glands of the rest of the body. The glands when enlarged are isolated, seldom as large as a walnut, and cause no local irritation. Between gland and gland, the engorged lymphatic vessels rarely prevent any sign of hardness. It would appear that these glandular swellings precede the appearance of other symptoms.

The patient falls into a profound state of weakness, not even responding to any exterior excitement. The sense of touch is maintained in normal condition. The sense of pain is very acute; patients are able always to distinguish the sense of heat and cold.

The **muscular sense or conscious movement** is conserved almost wholly up to the last. The patients with their eyes bandaged have perfect knowledge of the different parts of the body experimented on. There are no subjective perturbations of sensibility, except the general cutaneous itching of the trunk and members which we believe to be independent of any eruptions. In the course of the disease no alteration of any note in hearing, smell, or taste was observed.

The **reflex sensibility**, both superficial or deep, during the sleeping period presents a variety of different combinations; when nervous depression was deferred the reflex action was increased. In the ultimate phases of the disease, however, the reflex sensibility is suppressed. In no case was the Argyll-Robertson pupil noticed. The reflex sensibility of the conjunctiva usually continued till the death stupor.

In some cases, notwithstanding the advanced stage of the disease, there did not exist any notable reflex disturbances, either superficial or deep. Muscular tremblings were noticed in all the patients. These movements begin by isolated contractions of the fibres of the muscles here and there without provoking movement of the members, then they become more accentuated, causing contractions of an entire muscle. At last the tremblings cause rhythmic oscillations of the limbs, the trunk and the features.

In the first phases of the disease all the movements are precise and co-ordinate. By degrees the movements become impeded and the walk slower. At last the "myasthenia" reaches such a degree as to preclude the possibility of standing.

Romberg's sign is always absent. Paralysis does not exist, and when the patients cannot leave their beds on account of the muscular "asthenia," they nevertheless can change position in bed and scratch themselves.

Convulsions or intense contractions we never saw; but we noted a rigidity, a feeling of hardness in the muscles. This state of the muscles make the patients, however, gradually take up certain fixed positions; one of these was the bending of the legs over the thighs in a *convex manner*, and also the forearms over the arms and the hands over the forearms.

In two cases only did we come across Kernig's sign.

Sleep dominates the morbid symptomatology of the brain. It is one of the initial manifestations, which in the first place arrests the attention of the patient. At the beginning the tendency is easily overcome, but afterwards the patients go to sleep in the midst of their occupations. It is not rare to see them going to sleep in the middle of dinner, leaving the food unmasticated in the mouth.

The attacks of sleep are never progressive and regular. There are some days when this symptom is very marked, and others in which it is almost absent, but finally the patient settles down to a permanent and almost constant sleep.

It is never so profound that they cannot be wakened with relative facility, but then they fall to sleep again when they are left alone, and the sense of hunger wakens them.

In some acute cases, when the patients are prevented from doing anything, they attempt to bite the persons attending on them, crying out, and after falling into a paroxysm of tears become calm again. Others become melancholic and sad from the beginning of the disease, constantly weeping and in a marked state of physical depression. One woman had fits of gaiety and laughter followed by profound torpor and cerebral "asthenia."

The memory does not seem to be affected. The volition is evidently influenced by the sleep and as the disease increases so the will power becomes less.

The concentration of the attention becomes more difficult as the disease progresses. We have never noticed alterations of speech or in the manner of writing. We have never observed cases of "paraphasia," "jargonaphasia," or any other important alteration of this sort.

In the **circulatory apparatus**, the most notable

symptom, which is almost constant, is the diminution of the arterial tension. We have noted this symptom in patients in whom the disease was only beginning.

The pulse, frequently normal at the beginning, becomes in the more advanced states irregular and generally ends by being arrhythmic and thready. No cardiac bruit has been noted, but a modification in tone of the heart sounds is the rule.

The blood changes observed noted by Manson in two cases of "*filaria perstans*," we have never met with.

The percentage of hæmoglobin, measured by a hæmometer, of Herschel, is always of less degree, varying between $\frac{1}{10}$ per cent. and 10 per cent. We have not observed anything abnormal in the white and red corpuscles; from the contents of the corpuscles we cannot draw any definite conclusions.

The temperature chart of the patient never has a cyclic evolution. Now and again there are febrile accessions, generally in the evening; malarial parasites were only found in one case. The temperature rises usually just before death.

The lungs remain normal as a rule; in one or two instances some hypostatic congestion was observed. The rhythm of the breathing is maintained until towards the end, when slightly marked Cheyne and Stokes respiration has been observed. No "pituitary" signs or symptoms were present in the nose, nor any marked affections of the pharynx, gums, or teeth. The tongue always showed itself as clammy and wet; profuse salivation we never saw; the tonsils appear normal. The patients preserve their appetite to the end.

In no cases was vomiting present, nor do we attach any specific importance to occasional attacks of diarrhoea. The liver and spleen are usually passively enlarged; jaundice does not occur in sleeping sickness.

The analysis of the urine made systematically shows constantly the presence of albumen in small quantities, in some cases only traces; no sediment is, as a rule, to be met with. The "urobilin" existed always in greater quantity than the normal in the urine allowed to stand. I have seen in some cases, without notable "polyuria" coinciding with a density relatively small, a notable diminution of urea and the phosphates.

In adults the power of erection of the penis is lost. It is an important fact that the *post-mortem* examinations reveal the constant presence of an infra-arachnoid secretion, noticeable in the ventricles and the "sub-serous" spaces. In some rapid cases the effusion of this liquid assumed immense proportions. The liquid is never transparent, always with more or less turbidity, sometimes bloody, but we have never seen it take the macroscopic character of pus. The presence of this secretion is evidently due to the existence of an inflammation—pia-arachnoid meningitis—of acute and variable intensity.

These lesions predominate on the convexity of the hemispheres; the cerebellum is also a favourite seat of this effusion. By these lesions the dura mater becomes adherent to the subjacent meninges, but adhesion of the pia mater to the cerebral substance does not exist.

When the disease becomes more acute the effusion becomes more abundant, with marked congestion of the arteries and the veins of the dura mater without the formation of adhesions; effusions occur into the ventricles; a few adhesions and very occasionally some softening of the thalamus opticus and adjacent part of the brain tissue are met with.

In the cerebellum occasionally there is a predominance of inflammation in the upper "vermis." Similar changes to those observed in the cerebrum have been observed in the cerebellum.

In the heart, the only morbid changes which appear constantly are the yellow colour of the surface of the heart, and concentric hypertrophy of the left ventricle. There never are any signs of endocarditis. Lesions of the pericardium I have seen a few times only. Apart from old pleuritic adhesions, there was nothing found of an abnormal character in the respiratory apparatus; in the lung tissue pneumonic congestion was found once or twice.

The glands at the bifurcation of the trachea were frequently enlarged.

The spleen varies in volume, being diminished or increased; it usually becomes of a leaden colour and its consistence is but little changed.

The liver does not usually present any macroscopic differences from the normal state.

The kidneys have sometimes signs of congestion and also slight inflammation.

In the other viscera nothing was found worthy of note. The lymphatic abdominal glands were generally swollen.

(To be continued.)

THE DURATION OF THE LATENCY OF MALARIA AFTER PRIMARY INFECTION, AS PROVED BY TERTIAN OR QUARTAN PERIODICITY, OR DEMONSTRATION OF THE PARASITE IN THE BLOOD.

By DR. ATTILIO CACCINI.

Assistant Physician, Hospital of Santo Spirito in Sassia, Rome; Lancisi Clinique, under the direction of Prof. Giulio Bastianelli.

(Translated from the Italian by St. Clair Thomson, M.D. Lond., F.R.C.S.)

PART II.

ON RELAPSES AT SHORT INTERVALS.

(Continued from page 155.)

(2) QUARTAN WITHOUT QUININE TREATMENT.

As a rule, in every quartan case in which we did not exhibit quinine, we would repeat what we stated in reference to spring tertians. That is, after a greater or lesser number of attacks more or less typical, the febrile attacks did not return. This result obtains whether the patient continues to work, or whether he is under observation at the hospital leading a life of strict repose in bed, with a rigorous hygienic diet, and with abundant and healthy nourishment; in the latter instance, as a matter of fact, the fever ceases in a shorter time and in a greater number of patients. In similar conditions this spontaneous

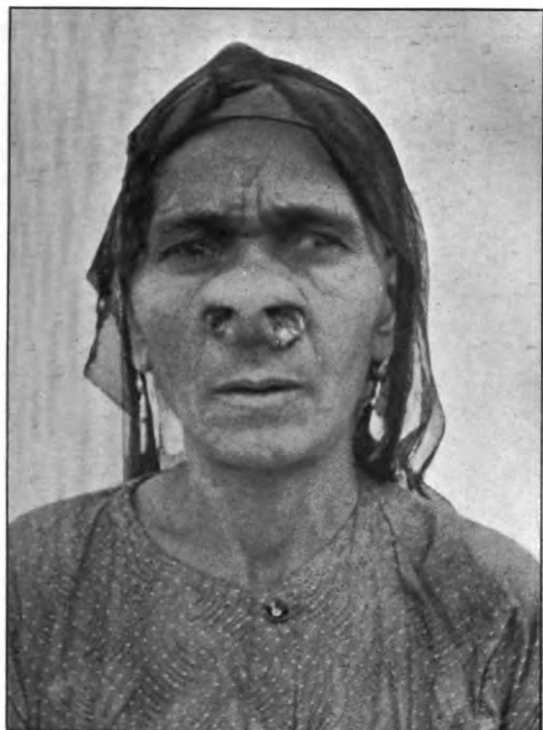


FIG. 1.

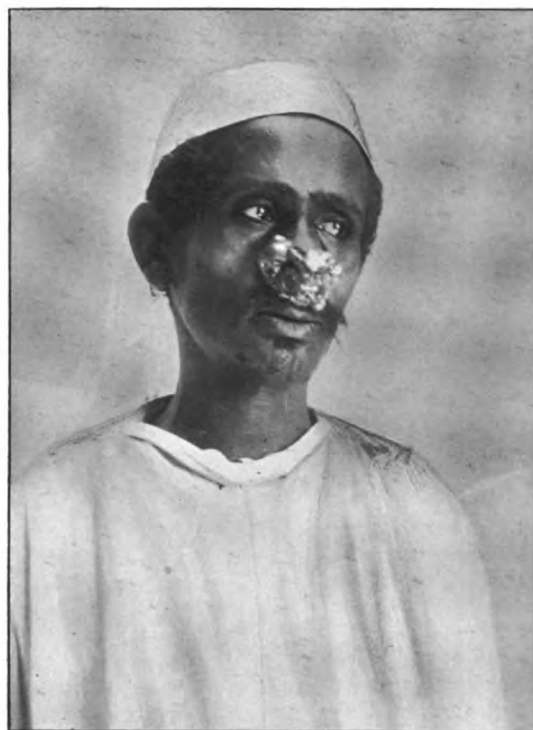


FIG. 2.



FIG. 3.



FIG. 4.

Same as fig. 3, showing the non-implication of the edge of the upper lip.

PHOTOGRAPHS ILLUSTRATING CASES OF SARCOMA OF THE NOSE.

By FRANK COLE MADDEN, M.B., B.S., F.R.C.S., Professor of Surgery, Egyptian Government School of Medicine, Cairo.

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for

disappearance of the primary infection is more prompt even in the spring tertians than in the tertian quartans. We may note also that while relapse occurs without our detecting the determining cause in the spring tertians, in the quartans not submitted to quinine, and in similar conditions, the attacks do not continue to manifest themselves except as a consequence of determining causes, which we can establish exactly in the greater number of patients. Hence, there is definite and diverse behaviour between the spring tertians and quartans; in fact, all the quartans observed for a certain time in the hospital never had a relapse at short interval if they were not exposed to some of the accidental determining factors of which we spoke at the beginning; while in the spring tertians the relapse, as we have seen, occurred within a period of time varying between five and eighteen days, and independent of accidental factors. These factors in the spring tertians have no other effect than to abbreviate the duration of the latent period; and it is for this reason that patients left to themselves relapse after a shorter apyrexial period than in the patients well treated, in whom it is easy for the apyrexial period to reach the maximum limit of eighteen days. These facts, met with in patients in the hospital, were also confirmed every time an observation was made on an individual who lived in almost identical conditions to those of patients in the wards. On the disappearance of the febrile attacks in the quartan cases there was constantly a corresponding improvement of the general condition and of the anæmic. This improvement was certainly rapid, so much so that the patient, deceived by his sense of well-being, often wished to take his discharge. *I was never able to note any case of complete spontaneous cure of quartan infection; the relapse came either in a short interval or after a month or so, that is to say, as soon as the patient was no longer in hygienic conditions with regular diet and rest.*

This I was able to show in 118 cases of quartan infection which I was able to follow for a long time without submitting them to the quinine treatment. Indeed, in these 118 cases only 8 relapsed after a short interval, and only in consequence of one of the usual factors (see Table).

(3) OBSERVATIONS.

One can, however, assert that individuals of any condition, age, profession, or station, when compelled to lead a regular life of rest, removed from fatigue and exposure, supervised in their diet, not exposed to damp, wet, cold, &c., can remain for a long time in conditions of good health without any relapse at short interval; while, on the other hand, if any of these patients did not follow this method of life even for a short period of time, they always inexorably met with a relapse, sometimes even after a few hours. However, while in the spring tertians, as we have seen, the neglect of the quinine treatment infallibly leads to relapse at short interval (only one escaped this out of 121), in the quartans, on the other hand, either with or without quinine they managed to escape this relapse, providing that they avoided the accidental factors of relapse.

Another statement that can be made is that while in the case of the spring tertian the patient, though improving, never returns to complete health (indeed he has both general and local disturbances during the apyrexial period); in the quartan, on the other hand, this cure is absolutely complete, and the patient does not complain of any special disturbance during the apyrexial period. One can even show, by objective examination, that the alterations of the general condition diminish until they cease entirely and we have complete reduction of the splenic tumour, which, on the other hand, in spring tertian, constantly lasts into the apyrexial period with very little alteration. This, let us repeat, is in the case of patients who are well looked after.

We may, however, from that conclude that there probably is no true and proper relapse at short interval unless the patient exposes himself to some of the accidental factors of relapse.

It may be worth recalling once again the relapse which took place in 8 of the 118 cases of quartan not submitted to quinine, as soon as they were exposed to the action of one of the mentioned factors.

Now it results from daily observation, that quartan infection has a character of great obstinacy, and that a sequela of relapses at short interval almost constantly occurs with it. Well, now, that in no way contradicts my observations, inasmuch as it is easy to understand that it is very difficult for these quartans to lead a life so hygienic as to avoid all the accidental factors of relapse. On the other hand, this leads to another affirmation, and that is, that the quartan infection has a special obstinacy, owing to which, whether treated or not, it remains latent for an indefinite time, always ready, however, to produce a febrile attack as soon as the individual offers the very smallest diminution of his organic resistance.

Hence, to induce a relapse a return to habitual work, even though not fatiguing, may be enough. On the other hand, admittance to the hospital is in itself sufficient to cut short a relapse. And since the examination of the blood of quartans is almost always negative immediately after the outbreak of relapse, these quartans are often taken for malingerers, since, when the medical visit takes place, they have already had sufficient rest to disperse all the superficial symptoms of the disease, and the microscopical examination of the blood has been omitted.

Is it then possible to establish the duration of the latency of quartan?

On the whole we may say that the relapse at short interval, while it occurs in the spring tertian within certain limits, and often in spite of any precaution, in the quartan, on the other hand, the relapse at short interval is always avoided when the patient is kept in regular conditions of hygiene and diet. But as soon as these cease the relapse occurs quickly and in spite of treatment after no matter what lapse of time, since we were able to prove a relapse even after only four days of apyrexia in a patient who, as soon as he left the hospital, was exposed to rain for some time (five hours).

It seems here opportune to recall what took place in all the cases of inoculation of quartan which I

attempted for purposes of study. I was able to protract at my pleasure the period of incubation, by keeping the patients whom I had inoculated in good hygienic conditions (that is, insisting on rest in bed, good food, &c.), without giving them any medicine whatever. On the other hand, I was able to abbreviate this period by exposing the patients to cold baths and the application of electricity, mustard leaves, &c., in the region of the spleen. So I was able in some cases to make an incubation period of more than ninety days, and in others of seven to eight days. It is worth noting that the febrile attacks only appeared when I wished them, and that was when I exposed the patient to one of the said debilitating causes. A similar condition held good in the relapses.

(4) SUMMARY.

Summarising the facts stated we come to the following conclusions:—

That quartan is the most obstinate in relapsing in reference to relapse at short interval, and in frequency. Many authors have already observed this. It would appear, however, that quartan only relapses in consequence of determining causes, which are nearly always traceable in each case. That is, quartan has not a true and proper period of latency for the relapse at short interval, but the duration of this period, instead of being constant and uniform, is determined by the intervention or non-intervention of a given occasional cause, after which the febrile outbreak follows shortly (eighteen to forty-eight hours). There is no preference between one or other of the causes of organic weakening, but probably the most important factors (and perhaps also the most frequent) are rain, cold, traumatism, &c.

The relapsing febrile attacks, typical or not, can appear even after a period of two months of latency, on the intervention of any accidental factor. It only remains to see whether in quartan also the outbreak of infection is always subordinate to the intervention and effect of a determining cause, as always happens with the outbreak of a relapse, at least as I have generally remarked. It is necessary to see if, during the long period of incubation, which it is determined may last two months,¹ there exist endoglobular forms circulating in the blood, and following a cycle of constant development of seventy-two hours, without any manifestation. On the other hand, I have never, up to the present, had any opportunity of observing this. Besides, we may say that either in the internal organs, or perhaps even in the circulating blood, there is in some shape, or even in shapes not up to the present studied, the germs of the disease for which it only requires an occasional cause to give rise to their development in the known forms of fever-producing cyclical endoglobular parasites.

The intervention of the determining cause is not difficult when one has to do with individuals who live and work in the Campagna, badly clothed, lodged, and fed, subject to irregularities, and to physical and mental traumatism, so that it is easy after twenty-five to thirty days' residence in malarial quarters for the

quartan to break out amongst workers. Also, it is not rare to meet with cases in persons in our hospitals who although removed for some time (two to three months) from malarial districts without ever having had any symptoms, yet in consequence of a traumatism, an administration of chloroform, some intercurrent illness, cold, or change of air, are seized with quartan febrile attacks, with positive results from the examination of the blood. In these cases one is forced to admit that the infection has existed for a long time without ever having had the opportunity of breaking out. In fact, whether for the incubation of the primary infection or for the latency for the relapses, there is probably no fixed period, both the incubation and the latency ceasing only on the presence of some accidental factor.

Just as in quartan there does not exist a true and exact fixed and determined period of incubation (and this results from the observations carried out on patients inoculated by me with quartan, as I have shown above, and from what authors have here and there recorded), so for quartan itself there appears to exist neither a fixed and determined period of latency after the exhaustion of the fever or its disappearance under quinine. This fact can be demonstrated almost constantly when with careful observation and with minute interrogations of the patient, one succeeds in exactly weighing every minute detail of the history. Quartan fever, while in appearance distinguished from spring tertian, and as we shall see from malignant tertian, because it has not like these an exact period for the duration of incubation and latency, still by examining carefully one sees that it behaves in a similar manner to malignant tertian and to spring tertian. In fact in spring tertian and in malignant tertian the period of latency behaves in duration like that of incubation; and since this latter constantly oscillates within certain fixed limits, so also the latency oscillates in the same limits of time; while the quartan, which has no fixed duration of time for its incubation, has neither any for its latency.

CHAPTER 3.—MALIGNANT TERTIAN.

We know that in malignant tertian the apyrexia which follows, whether owing to the spontaneous exhaustion of the fever (exhaustion, however, which is known to be not at all rare), whether to the quinine treatment, is not as a rule enduring. My researches have shown that the apyrexia lasts as a rule from five to twenty-one days, and that the outbreak of the relapse is in no way impeded or retarded by the quinine treatment, whether in itself or combined with arsenic and iron. These facts besides, are found noted here and there by various authors. . . .

(1) CASES OF MALIGNANT TERTIAN TREATED WITH QUININE.

It is well to recall that it is not suitable to carry out a comparison between various individuals and the various systems of quinine administration already stated in regard to spring tertian and quartan. Because, given the danger to which they are exposed in summer fevers, when left without quinine, one is as a rule compelled to administer the remedy during any

¹ "Proceedings of the Society for the Study of Malaria," 1901, vol. ii., p. 108.

period of the fever, or of the apyrexia, as soon as ever the examination of the blood, or serious symptoms, show that we are in the presence of a summer malarial infection.

However, from the patients that we were able to observe for some time, in twenty of the most robust and in whom one was able with confidence to await the opportune moment, the treatment was attempted of administering the quinine shortly before the febrile outbreak; in twenty-three other patients at the acme of the fever, and in nineteen others during the sweating stage. Now as to the result: Except for the modifications in the temperature curve induced by the treatment (modifications already excellently stated by Marchiafava and by Bignami in their classic work on "Spring and Autumn Fevers"), nothing was met with differing in the duration of the latency of the infection; i.e., the fever appeared always in all the three categories of cases between the fifth and twenty-first days, maintaining almost the same proportional numbers of relapses as in the patients who were not submitted to quinine.

It is to be noted that all the patients with malignant tertian were energetically treated with quinine, being given a gramme and a half or two grammes of quinine, per day, even after the cessation of febrile outbreaks. This administration was carried out according to occasion either by the mouth, the rectum, hypodermic or intramuscular injections, intravenous, or by a mixed method. Some days the dose was often larger than usual, reaching three or four grammes in twenty-four hours. However, the relapse occurred all the same, and in spite of the energetic treatment it occurred invariably between the fifth and twenty-first day of apyrexia. We were able to observe that as a rule, an equal period of time did not elapse between one relapse and another, in the same individual. Only in one case had the fever through these relapses regularly returned in the early hours of the eighteenth day of apyrexia, seizing the patient constantly while in a condition of the best *bien-être*. In this case the attacks were of a pernicious and delirious form, and in spite of the quinine, lasted one or two days. In almost all the other cases of spring tertian in which quinine was administered a few hours after admission to hospital, without it being possible to establish exactly in what moment the patient was fully "quinined" (there were 3,607 cases), it was possible in a majority of instances (i.e., in 2,003), to follow at least one relapse, and we were able to show that there was no notable difference in the percentage of relapses, between the patients who remained permanently in hospital during the apyrexia, and those who, deceived by the relative comfort of the apyrexia, asked for their discharge as soon as the fever ceased. I was able to show this by the study of a small group of workers (160 men and women) whom I followed for some time. All these workers fell ill late in July from various malarial infections, 90 of them from summer infections. All the 90 relapsed between the fifth and twenty-first day of apyrexia. It is worth noting that the whole group were regularly submitted to energetic treatment by quinine.

Of 792 patients whom I followed in the hospital for more than thirty days, all relapsed except 24 (we

must, however, note that these 24 patients did not lend themselves well to observation, either in consequence of their riotous character or from special conditions of health).

I was only able to show that the relapse occurred as a rule sooner in those patients who left the hospital as soon as the temperature fell, or in those workers under observation in the farms, than in the patients treated in hospital.

In the former case, indeed, apyrexia lasted from five to nine days, and only a few relapsed between the ninth and eighteenth day of apyrexia; a good number only relapsed between the seventeenth and twenty-first day, and relatively few relapsed between the ninth and tenth days.

This, of course, was in patients who continued the quinine treatment at the dose of one to two grammes per day.

I did not find any sensible difference in the duration of latency in reference to sex or to age. The greater number of relapses in adults is only due to the smaller proportion of women and children who engaged in work. Still I would say in parenthesis, in children, one or more relapse of summer malaria may pass unobserved, and, as a rule, is often followed by a serious outbreak of unexpected pernicious fever. On the other hand, a remarkable influence on the outbreak of relapse is produced by scarce or bad nourishment, rain, moisture, cold, traumatism, excessive fatigue, gastro-intestinal disorders, &c., which shorten in a remarkable way the period of latency in the relapses at long interval. Indeed, in the fields, when a certain number of workers have been exposed to these causes, even after a few hours, but as a rule after one or two days, there is an outbreak of relapses. Besides it is known and proved in reference to the period of incubation which is considerably abbreviated in those who, being still infected, are exposed to the effect of one of those causes of general debility.

That exposure to these causes suffices to shorten the period of latency is proved by the simple fact that a wet, cold, hot, or damp day is not followed by any shortening of the period of latency in patients under treatment in the hospital; with these it is found that the attacks recur at the regular interval and not sooner than usual. The case of the hospital patients is almost similar to that of landlords and proprietors who are generally well sheltered; in these the attacks do not recur except when they have been directly exposed to the aforesaid debilitating conditions, while labourers, on the contrary, are subject to continual attacks. It has been noted that numerous cases come to the hospital for treatment during rainy, cold, and damp weather, especially when such weather comes on suddenly, while in the patients who have been some time in the hospital, well nourished, clothed and sheltered, in short, not exposed to inclement changes, the attacks are the same as in normal weather. I have also noticed that every time a sudden change of weather has caught the convalescent patients in the garden during the hour of recreation, there has been a considerable and rapid increase among all the patients exposed to the said change of weather, though only for a short time. The same thing occurs among the landlords and proprietors; if, as an exception, they

expose themselves to any of these debilitating causes, the period of latency is then considerably shortened, varying from five to nine days as among the labourers; and this in spite of their nourishment being very much better than that of poor labourers, and in spite of their being better able to follow the quinine treatment, their easier circumstances enabling them to call in medical assistance more easily than the poor labourer.

(2) CASES OF MALIGNANT TERTIAN NOT TREATED WITH QUININE.

My observations with regard to attacks of summer malaria not treated with quinine have been few, as the severity and aggravation of the symptoms called for the most energetic therapeutical intervention. I may say, however, that upon 3,704 cases I was able to follow 44 who had never made use of quinine, and these patients suffered the attacks although they were in the hospital, far from the common predisposing and determining causes. All suffered attacks within a period varying from five to eleven days, as appears from the following table.

Duration of latency (in days) in cases of summer malaria never treated with quinine:—

| CASES. | DURATION OF APYREXIA. |
|----------|--------------------------|
| 9 | 5 |
| 1 | 6 |
| 5 | 7 |
| 1 | 1 |
| 7 | 9 |
| 1 | 10 |
| 1 | 11 |
| 1 | 12 |
| 1 | 13 |
| 1 | 14 |
| 1 | 15 |
| 1 | 16 |
| 2 | 17 |
| 6 | 18 |
| 1 | 19 |
| 3 | 20 |
| 2 | 21 |

Total, 44 patients who had never used quinine.

From the table it appears that the duration of latency in summer malaria in patients not treated with quinine varies from five to twenty-one days, and that the attack recurs most frequently in a descending line, on the fifth, ninth, eighteenth, seventh and twentieth day of apyrexia, while the intervening days show a minimum. From the observation of these cases it may be gathered that the period of latency is not of uniform duration in every individual. Indeed, out of fifteen patients who were under observation for more than two attacks, in only one did the attacks recur constantly on the ninth day of apyrexia, and this in six successive attacks.

CHAPTER 4.—CONCLUSIONS.

(A) *Malignant Tertian*.—The duration of latency in shortly recurring attacks varies from a minimum of five to a maximum of nine days, which is also the usual duration of the period of incubation. We

further observe that there is another maximum of attacks on the sixteenth and eighteenth day, but in such cases the attacks or the development of the parasite may also have occurred towards the ninth day, but with symptoms and biological phenomena which are not open to observation.

This occurs even in spite of the quinine treatment.

(B) *Spring Tertian*.—The period of latency in shortly recurring attacks is from five to eighteen days, which is about the period of incubation.

The accidental factors mentioned elsewhere affect and shorten the period of latency.

(To be continued.)

THE SIGNIFICANCE OF BLUE PIGMENT SPOTS.—Dr. Baelz, of Tokio, seems to have made a discovery of great interest in anthropology. Others before him had noticed the blue spots which Japanese babies have on the lower part of their spine and elsewhere, and which usually disappear before the age of 6, but no one before him, so far as is known, had interpreted and set forth this phenomenon as a peculiarity of the yellow race in contradistinction to the white race. In Korean and Chinese children, in Malays and Eskimos, these blue spots have also been found. They are not visible on European children, but the pigment cells have been found microscopically. Dr. Baelz says: "The white, the yellow, and the black man have all the same kind of colouring matter in their skins; the difference is only a matter of degree. In the white the pigment does not show, or hardly shows at all, to the naked eye; in the yellow it is a little more abundant, hence the yellow tint; and the more abundant it is, the darker the skin will look until we reach the negro." In Euro-Japanese children, if the offspring resembles the fair-haired, blue-eyed parent, it has no spots at all; if the influence of the Japanese and the foreign parent is about equal, the spots are there though more or less indistinct. But if the Japanese characteristics prevail generally, the spots are almost as well marked as in a Japanese baby. While apes have the same blue spots, and certain monkeys blue callosities on the buttocks, Japanese children have sometimes one-half of their bodies covered with them. Dr. Baelz believes that the blue spots form a most important racial characteristic, and will not acknowledge that the configuration of the eyelids or of the earlobes is of anything like equal value.—*The Nation*.

THE scientific expedition being sent out by the London School of Tropical Medicine to Uganda to study sleeping sickness will start in a few days. In our notice of this expedition Dr. Low's name was printed in error as Dr. Lord. Dr. Low holds the Craggs Scholarship of the London School of Tropical Medicine, and his previous record of work at home and abroad testifies to the wisdom of selecting him to take charge of an expedition of the kind.

ANTI-TYPHOID INOCULATION.—The reply given in the House of Commons to enquiries concerning the results of anti-typhoid inoculation was that a report of under 5,000 cases had been received, but that the results were not sufficiently conclusive to justify any official statement on the treatment.

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THE

Journal of Tropical Medicine

JUNE 2, 1902.

POPPY CULTIVATION IN INDIA.

THAT most indefatigable body, the Society for the Suppression of the Opium Trade, has again been showing signs of activity. In December, 1901, the Society addressed a memorial to the Prime Minister, calling his attention to the policy followed by the Indian Government of late years of largely extending the cultivation of the poppy in British India, notwithstanding numerous and express pledges on behalf of successive Governments in this country that no such increase should take place, but that, on the contrary, this cultivation should be diminished.

The memorial was also sent to the Secretary of State for India.

The Society in specific terms accuses the British and Indian Governments of deliberately neglecting their promises. A grave charge, and one which not only British statesmen naturally

resent, but which British people are not likely to accept on mere statement from any Society, however well intentioned.

The reply to this assertion, given by Lord George Hamilton, the present Secretary of State for India, is at once definite and conclusive, namely, that he "cannot admit that there is any pledge of this kind which is operative at the present time or binding on the Government."

We are, as a people, accustomed to rely on the word and pledges of our statesmen, and we are not prepared to throw over the traditional good faith of our rulers. The members of the Society seem, however, to take for granted that these high officials are not speaking the truth, and expect the public to believe their word as against cabinet ministers.

What is it that the Society wants to do? There are two distinct questions mixed up in their demands. One is that, by recognising the opium traffic, the Government of India and the British people generally are guilty of a "national sin," by deriving revenue from an article which is poisoning the Chinese. The second factor in the argument is the harm that is being done to the Chinese as a people, by the consumption of opium. These are two wholly separate problems. Government can cease the exportation of opium to China, but by so doing can we save the people of China from being "poisoned" by opium? We cannot prevent the consumption of opium in China by stopping the supply from India. The Chinese are cultivating the poppy extensively, and it is only a question of time how soon the Indian supply will cease from the want of demand.

The Indian Government, being therefore incapable of dealing with the growth or consumption of opium in China, is therefore only guilty of the "national sin" of producing and trafficking in a deleterious commodity. If governments are to be held guilty on such grounds, then is every government past and present to be attainted on such purist terms.

Any government deriving a penny of income from any wrongly used substance or article which affects the national health is guilty of a "national

sin" when adjudged by such a tribunal. Alcohol on this plea should go duty free if a government is to remain guiltless; every person who commits suicide or injures another in anyway, by any of the poisonous vegetables or metals used in the arts from which government derives income by taxation, can lay the blame on the individuals who hold the reins of government for the time being for allowing the use of such materials. Granted such premises, then mankind is the sinner, for all peoples that dwell on the earth come under the ban.

If the Society is struggling to lessen the amount of opium consumed by the Chinese or any other race of people, they will have not only the Chinese themselves, but all civilised and uncivilised communities with them. If, however, they are attempting to set up a code of national morals based on the opium exportation from India alone, they cannot expect public support. The loophole given the Society at the present moment is the fact that during a period of excessive supply (1888), an unusual stock of opium caused the government of India to restrict the area of poppy cultivation, and during the seven subsequent years, owing to bad crops, &c., the quantity continued below the average. Afterwards, when the market improved and the crops gave better yield, the quantity exported again reached the normal average. It is this apparent increase which gave the Society for the Suppression of the Opium Trade an opportunity of publicly accusing the Governments of Great Britain and India of insincerity. The interference may be opportune, the motive may be good, and we have no doubt as to the purity of the humanitarian principles involved, but we do wish to have this question discussed on a rational basis and not to make it a subject of mere acrimonious discussion; one in fact in which Government officials are flouted as consummate liars.

Can we save the Chinese as a people from the deleterious influences of opium is a question which all right-minded people will be willing to take part in. It is not to be done by stopping the supply from India. The quantity sent from India to China is a mere bagatelle to the actual

quantity grown and consumed in China. Nor can we by accusing our rulers help the Chinese. Other methods must be tried. What these may be cannot be settled off hand, but did the Society, which has devoted so much pains to injure the reputation of the officials of their own people by publicly accusing them of falsehood, turn their attention to the real evil, and direct our energies to lessening the deleterious influence opium causes in China, they will have the support of every right-minded person. The "national sin" may be with us, but the physical and moral harm is to the Chinese, and the two questions have no direct or indirect bearing.

Translations.

GEOGRAPHY AND HISTORY OF LEPROSY IN CRETE.

By DR. EHLERS (Copenhagen).

Abridged translation from the French, by P. Falcke.

THE island of Crete is considered to be one of the prettiest of the Mediterranean islands, and although possessing a chain of lofty mountains it has no large water-courses. The water is supplied by numerous torrents from the mountains, which are fed by the torrential rains and by melting snow. These torrents water the plains during the winter but run quite dry during the summer.

Pits and springs provide potable water to the inhabitants of the plains, while the residents of the mountains are forced to store rain water in cisterns or to use snow water.

The climate of Crete is much cooler than that of Cyprus, Malta and Algiers, though in the same latitude.

THE SANITARY ENVIRONMENTS.

The people of the towns live in dark lanes, so narrow that the sun is rarely seen, and most of the houses, dating back to the time of the Venetians, do not by any means conform in hygiene to the requirements of civilisation.

The population of the villages are still more disadvantageously situated as regards sanitation. Their dwellings, most of which are in the centre of plantations, are low, dark, dirty and damp. It is almost impossible to obtain fresh meat and the diet of the villagers is almost exclusively vegetable, consisting of ripe fruits, vegetables, rye bread, a quantity of oil, and a sort of brandy made of the dregs of wine (Raki).

In consequence of these unhealthy conditions the people are far from healthy and suffer greatly from the effects of endemic malaria, which is particularly prevalent in the valley of Rethymno and on the plain

of La Canée. They are also subject to *leprosy*, which has been endemic on the island from time immemorial. It is impossible to trace back the genesis of leprosy on the island, but the disease was probably a heritage from the Phœnicians; for Galène, Ætius and Celse unite in designating it as "the disease of the Phœnicians."

DATA AS TO THE NUMBER OF LEPEERS.

The first author who mentioned leprosy in Crete was Savary (1779), who expresses himself on the subject as follows:—

"This beautiful country is cursed with a disease which, though less dangerous to life than *plague*, is more repulsive. Syria was the ancient centre of *leprosy*, and was thence communicated to several islands of the Archipelago. The disease is contagious, and is immediately transmissible by the touch. Lepers are relegated to little huts built by the wayside, and are prohibited from leaving them and communicating with anybody. They usually have a little piece of ground where they cultivate vegetables and keep fowls; by these means and the charity of passers-by they drag out a miserable existence.

Rich persons are not attacked by this disease; the lower classes, and more particularly the Greeks, are most subject. The Greeks strictly observe the four fasts, and during the whole of these periods they eat nothing but salted fish, *boutargue* (fish roe salted and smoked), olives preserved in brine, and cheese. They also drink a large quantity of the native coarse, hot wines. I am of opinion that the reason the disease is not found amongst the well-to-do Turks, nor amongst the Greeks living on the mountains, is that the former eat meat, rice and vegetables throughout the year, and the latter eat principally fruit, vegetables and milk foods."

Sieber, the botanist, 1814, also mentions leprosy. He first saw the disease at the fort of Candia, where a suburb was put apart for their occupation, and they were prohibited from entering the town, and this was the custom in the three largest towns on the island. At that time leprosy was so prevalent that there were one or several lepers in each of the 600 or 700 villages. They were permitted to live in common with other persons until their skin was covered with eschars and their fingers began to fall off, when they were sent to the "*leprochorion*" (leper village).

Captain Spratt (acting Admiral of the English Navy) was the next person to study leprosy in Crete (1851 to 1853). He mentions that at that time 111 lepers, members of 70 families, of which 32 were Mussulmans, lived in the leper suburb of Candia. He describes their manner of living, and says that the population believed that the disease was contagious. There were 100 lepers in the leper village outside Hierapetra. Dr. Smart, who was probably the medical officer on board Captain Spratt's ship, mentions that the total number of lepers on Crete, according to his computation, was at least 900.

Dr. Hjorth (1816 to 1861), the principal sanitary officer of Crete, expresses himself as follows: "Leprosy attacks a relatively large number of the population.

It is difficult to indicate the initial cause, the inhabitants of various districts living in almost an identical manner; but the greater part of the lepers live in the mountains of Sphakia and Selino, where snow water is almost exclusively drunk." Hjorth gives the total number of lepers in the island in 1853 as 578.

Dr. Brunelli, in an important work on leprosy in Crete, computes that in 1863 there were only 400 lepers on the island.

LIABILITY ACCORDING TO RACE AND DIET.

Dr. Varoncha, of Canée, who practised medicine in Crete for fifty years, observes the Mussulmen are less subject to the disease than Christians, and that some localities are notorious leprosy centres, while other places quite near by are immune. His conclusions summed up are as follows: "The disease is not contagious at first, he therefore does not consider it necessary to isolate the victims till at a more advanced stage. The patients should be nursed by old persons, who are not so liable to be infected. He is of opinion that it is dangerous to have intercourse with lepers suffering from diarrhoea or ulcers. The patients should be taught to follow hygienic principles, which they unconsciously neglect."

Dr. Varoncha, moreover, believes that the disease is extending on the island, a circumstance that is due to the personal uncleanness of the Christian peasants, as also to their abuse of wine and "*raki*" (brandy), and their excessive use of pork, which they eat fried in rancid oil.—(*Leprosy*, Bibliotheca internationalis, 1901, vol. ii., No. 3.)

Current Literature.

ASSOCIATION OF AMERICAN PHYSICIANS

Seventeenth Annual Meeting, held in Washington, D. C., April 29th and 30th, 1902. J. C. WILSON, M.D., of Philadelphia, President.

Drs. M. J. LEWIS and F. A. PACKARD, of Philadelphia, presented a report of the cases of thermic fever treated at the Pennsylvania Hospital in the summer of 1901. The total number treated was 91, of which 31 cases were women and 60 men. In 50 per cent. of the cases there was an alcoholic history. In many cases abstraction of blood had been practised, the quantity withdrawn varying from seven to twenty ounces. In no instance did it do any harm, and in many instances it was followed by marked improvement. Saline infusion into the median basilic vein proved useful in some instances.

Dr. Frank BILLINGS, of Chicago, cited a case in which the temperature, on admission to hospital, had been too high to be measured by the ordinary clinical thermometer. After a cold bath the man had gone into collapse, but had been revived, and then the temperature had quickly risen from 100° to 110°F. The man had eventually recovered.

Dr. J. H. MUSSEY said that of six cases having a temperature between 109° and 115°F., two recovered, and in these saline solution had been injected.

Dr. F. A. PACKARD, of Philadelphia, said it was the

opinion of all of those who had seen the saline solution used that more persons had been saved by this procedure than would have been saved without it.

LEPER COLONY IN THE PHILIPPINES.—The island of Kulion, distant from Manila about twenty hours' sail by steamer, was recently visited by the Commissioner of Public Health and the Sanitary Engineer of the Philippines Commission, to perfect arrangements for the establishment of a leper colony thereon. The island is about twenty miles long, ten miles broad, and contains many fertile valleys suitable for agricultural purposes. It is also well watered and timbered, and well adapted to stock raising. It is the intention of the Commission that the colony, after its establishment, shall be self-supporting. It is expected that about 600 lepers will be established on the island before the 1st of April, though the thorough carrying out of the plan as contemplated will require a number of years.—*Boston Medical and Surgical Journal*, March 6th, 1902.

PLAGUE.

PREVALENCE OF THE DISEASE.

INDIA.—During the weeks ending April 26th and May 3rd, the number of deaths in India from plague amounted to 17,302 and 15,748 respectively. The cities and districts chiefly affected were: City of Bombay with 589 and 486 deaths from plague; Bombay districts, 1,673 and 930; Calcutta, 577 and 441; Bengal districts, 553 and 473; United Provinces, 806 and 718; in the Punjab, 12,311 and 12,334.

EGYPT.—During the weeks ending May 11th and 18th the fresh cases of plague numbered 29 and 20, and the deaths from the disease, 12 and 13, respectively.

CAPE OF GOOD HOPE.—In Port Elizabeth, during the weeks ending April 19th and 26th, and May 3rd, the fresh cases of plague numbered 1, 3, and 0; there were no deaths from the disease. Port Elizabeth is the only town or district in Cape Colony infected by plague.

HONG KONG.—During the weeks ending May 17th and 24th the fresh cases of plague numbered 31 and 33, and the deaths from the disease, 34 and 33, respectively.

MADAGASCAR.—On May 27th, 8 cases of plague were reported at Majunga, and 4 deaths from the disease during the preceding week.

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Archives Russes de Pathologie, de Médec. Clinique et de Bacteriologie.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Brooklyn Medical Journal.

Caducée.
Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Esercito
Hong Kong Telegraph.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
Journal of the American Medical Association.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Medical Record.
Medical Review.
Merck's Archives.
New York Medical Journal.
New York Post-Graduate.
Pacific Medical Journal.
Polyclinic.
Public Health.
Revista de Medicina Tropical.
Revista Medica de S. Paulo.
Sei-i-Kwai Medical Journal.
The Hospital.
The Northumberland and Durham Medical Journal.
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The Journal of Tropical Medicine.

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Original Communications.

THE CLASSIFICATION OF THE ANOPHELINA.

By FRED. V. THEOBALD, M.A., F.E.S., &c.

THE Genus *Anopheles*, founded by Meigen in 1818, has now grown to a considerable size, about fifty species being known. Some of these present very close structural features, others very diverse ones. The genus as it now stands is becoming somewhat unwieldy, like *Culex*, and as there is seen to be such a diversity of characters in these Culices with long palpi in both sexes, I have been compelled to divide them up into several genera. There is a very great difference, for instance, between *A. maculipennis* and *A. pharænsis*, between *A. argyrotarsis* and *A. rossii*, the differences, in fact, are quite as great as between *Panoplitæ* and *Culex* or *Sabethes* and *Ædes*.

In the classification of the *Anophelina*, I have found, just as in the *Culicidæ* as a family, that the scale structure is the best to take for generic distinction. This grouping is based entirely on the scales of the thorax, abdomen and wings. It will be noticed that by these characters a natural grouping is formed, and that it, in the main, tallies with what we know of their larval structure. I do not think, however, that the minute structural differences in the larvæ should be taken as of greater value than specific characters; but it is of interest to find that, on the whole, by grouping by certain characters of the larvæ and certain adult characters we get similar results.

The following genera may be recognised :—

- Genus 1. *Anopheles*, Meigen.
- Genus 2. *Grassia*, nov. gen.
- Genus 3. *Cycloleppter*, Theobald.
- Genus 4. *Stethomyia*, nov. gen.
- Genus 5. *Howardia*, nov. gen.
- Genus 6. *Rossia*, nov. gen.
- Genus 7. *Leverania*, nov. gen.
- Genus 8. *Cellia*, nov. gen.

These genera may be tabulated as follows :—

| | | | |
|--|--|--|-----------------------|
| Thorax and abdomen with hairs only; palpi not densely scaled | Prothoracic lobes simple | Wing scales lanceolate | <i>Anopheles</i> . |
| | Prothoracic lobes mam-millated | Wing scales long and narrow | <i>Grassia</i> . |
| Thorax with narrow, curved scales; abdomen hairy | Wing scales small, lanceolate | Wing scales partly large and inflated | <i>Cycloleppter</i> . |
| | | Wing scales lanceolate | <i>Stethomyia</i> . |
| Thorax and abdomen with scales; palpi densely scaled | Abdominal scales on venter only; thoracic scales hair-like | Abdominal scales as lateral tufts and dorsal patches; thoracic narrow curved or spindle-shaped | <i>Howardia</i> . |
| | | | <i>Rossia</i> . |
| | | | <i>Leverania</i> . |
| | Abdomen completely scaled and with lateral tufts | | <i>Cellia</i> . |

Genus 1. *Anopheles*, Meigen (fig. 1).—Thorax and abdomen with hair-like curved scales, practically hairs; palpi in the ♀ thin, not densely scaled; wings with the veins covered with lanceolate scales, which may or may not form spots, which if present are never so numerous as in the other genera. The majority are large species.

Type: *maculipennis*, Meigen.

The following species are included in this genus besides the type: *bifurcatus*, L.; *walkeri*, Theo.; *punctipennis*, Say; *lindsayii*, Giles; *nigripes*, Staeger; *pseudopunctipennis*, Say; *stigmaticus*, Skuse (?). This genus was formed by Meigen on *maculipennis* and he included *bifurcatus*; later *nigripes* was added. I have therefore retained the genus in the restricted sense for these and allied species.

Genus 2. *Grassia*, nov. gen. (fig. 2).—Thorax and abdomen with hair-like scales; wings with long, thin lateral vein-scales; the wings are usually much spotted; the majority small or moderate-sized species.

Type: *rossii*, Giles.

The following are included in this genus: *superpictus*, Grassi; *funestus*, Giles; *rhodesiensis*, Theo.; *culicifacies*, Giles; *christophersi*, Theo.; *turkhudi*, Liston; *leptomeres*, Theo.; *gigas*, Giles; *cinereus*, Theo.

Genus 3. *Cycloleppter*, Theobald (fig. 3).—Differs from the two former genera, in that the wings have large inflated scales as well as typical lanceolate ones.

Type: *C. grabhamii*, Theo.

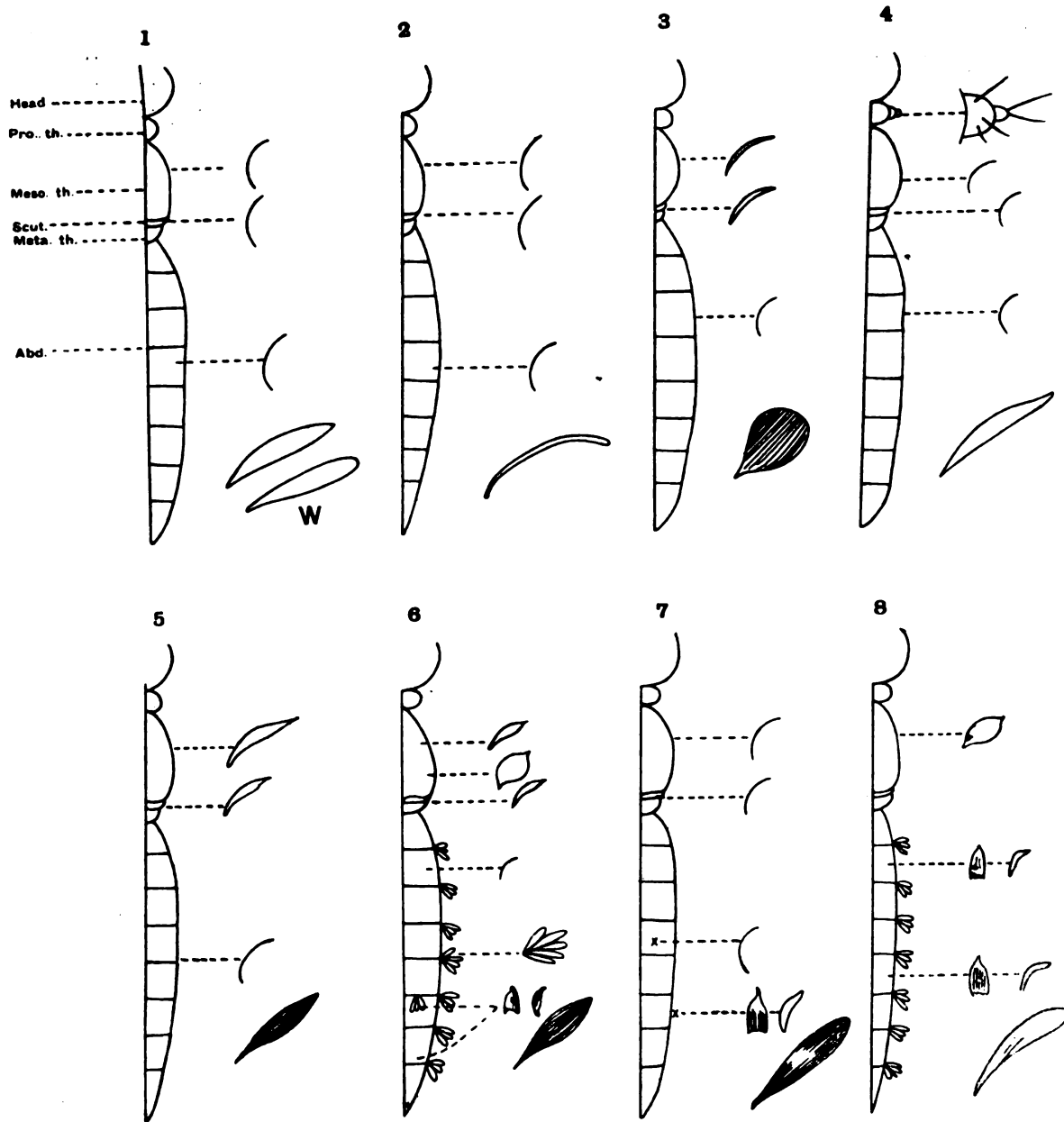
Two species only are known in this genus, the type and *C. mediopunctatus*, Lutz (ms).

Genus 4. *Stethomyia*, nov. gen. (fig. 4).—Thorax and abdomen hairy and bristly; prothoracic lobes mammillated; wings with narrow lanceolate scales, and the head with a median patch of flat scales. Palpi of the ♀ very thin.

Type: *S. nimbus* (n. sp.).

The genus contains only one species, readily separated by the head scales, mammillated prothoracic lobes and very thin palpi.

Genus 5. *Howardia*, nov. gen. (fig. 5).—Thorax with narrow curved scales; abdomen with hairs, no scales; wing-scales small and lanceolate; wings much spotted; palpi of the ♀ moderately scaled.



THE CLASSIFICATION OF THE ANOPHELINA.

1, *Anopheles*; 2, *Grassia*; 3, *Cycloleppter*; 4, *Stethomyia*; 5, *Howardia*; 6, *Laverania*; 7, *Rossia*; 8, *Cellia*.
(Showing general squamose characters.) W, Typical wing-scales.

Type: *costalis*, Low.

This genus includes besides the type, the following: *longipalpis*, Theo.; *minimus*, Theo.; *atratis*, Skuse (?).

Genus 6.—*Rossia*, nov. gen. (fig. 7).—Thorax with hair-like scales; the abdomen with ventral and apical scales; wing scales broadly lanceolate; palpi densely scaled in the ♀, and also the proboscis.

Type: *sinensis*, Wied.

This genus includes also all the sub-species of *sinensis* (*annularis*, V. der Wulp; *nigerrimus*, Giles; *indiensis*, Theo., and *pseudopictus*, Grassi); *barbirostris*, V. der Wulp; *paludis*, Theo.; *bancroftii*, Giles.

Genus 7. *Laverania*, nov. gen. (fig. 6).—Thorax with narrow curved and spindle-shaped scales; abdomen with lateral tufts of scales, with ventral scales and sometimes dorsal patches; wing-scales bluntly lanceolate; palpi densely scaled; legs mostly banded and spotted with white, the hind tarsi being often pure white.

Type: *argyrotarsis*, Rob. Desv.

This genus contains besides the type, the following: *argyrotarsis*, sub. sp. *albipes*, Theo.; *fuliginosus*, Giles; *jamesii*, Theo.; *maculata*, Theo.; *lutzi*, Theo.; *theobaldi*, Giles; *metaboles*, Theo.; *annulipes*, Wlk. (?); *masteri*, Skuse (?); *maculipalpis*, Giles; *kochii*, Donitz; *punctulatus*, Donitz; *leucophyrus*, Donitz (?).

Genus 8. *Cellia* nov. gen. (fig. 8).—Thorax with flat, spindle-shaped scales; abdomen entirely covered with scales and with dense lateral tufts; palpi of ♀ densely scaly; wing-scales large, bluntly lanceolate, wings densely scaled.

Type: *pharænsis*, Theo.

This genus also includes the following: *pulcherri-mus*, Theo.; *squamosus*, Theo.; *bigotii*, Theo.

PECULIAR MARKING OF THE TONGUE IN ANKYLOSTOMIASIS.

By PERCY H. DELAMERE, L.R.C.P., L.R.C.S.I.

Government Medical Officer, Leguan, British Guiana.

IN a letter addressed to Dr. Manson, Dr. Delamere writes: I am sending you an account of a symptom in ankylostomiasis, which I have observed in about fifty or sixty cases among the coolies on the two estates I have charge of in this island, as I do not see it mentioned in the books on the subject. I refer to a peculiar marking of the tongue. There is a large amount of ankylostomiasis in this island. I had fifty-one cases in one estate hospital during the twelve months from April, 1901, to March, 1902, and have eighteen cases under treatment now. Some little time back I began to notice that all the patients under treatment for this disease had a peculiar mark on the tongue, exactly as if the patient had just wiped a penful of Stephens' blue-black ink on his tongue; in fact, the first time I did take notice of it, was to ask the nurse why he let the patient play with the pens on the ward desk. The man said he had not, and that his tongue had been marked like that for a long time. A few days after, another man came in

a markedly anæmic state with a blue-black mark on his tongue; he also had ankylostomiasis. I then commenced to take notes, and found that all patients who had passed ankylostomes after treatment had more or less marked tongues. I even went further; we have a monthly inspection of new coolies on each estate during the first twelve months after arrival from India. I examined the tongues of every one of them and found fourteen who to look at were perfectly healthy; no anæmia, nor in fact any sign of disease, but who had blue-black marks on their tongues. Most of the cases said they had come from India with these marks; in fact, one man said his tongue was marked like that from childhood. I took them all into hospital and put them on the usual thymol treatment and every one of the fourteen passed mature ankylostomes. I now make it a rule that everyone with these marks on the tongue gets thymol even if no anæmia is present, and in no case have I failed to find ankylostomes. I have made an attempt to sketch and paint the tongues of the cases I have now under treatment in the hospitals, but please excuse the result, as I have no proper paint-box or colours, so have had to make the colour of tongue far too bright a pink, but the small patch of colour at the bottom of each group of tongues, I wish to represent the colour of a healthy tongue. The blue-black colour I have got more like what it should be, and the brown marks are like the colour of lightly-roasted coffee when first ground.¹

It must also be noted that the edges of the marks are not so sharply defined as they appear in my sketches, but fade away into the red of the tongue. I find that under treatment, thymol repeated two or three times, and large doses of tincture ferri. with a little quinine, that in from ten to twenty days the small marks have gradually faded away beginning from the edge and getting smaller. The large ones take six weeks or more.

I intend taking further notes, as if this is a regular symptom here of ankylostomiasis it is well worth making public, as it is an early sign I take it, before there is pronounced anæmia, and of such easy diagnosis that cases can be treated and cured before there is much damage done. I have had no opportunity of making sections of these patches.

FIG. 1.—Male, aged 28, taken into hospital on account of tongue, which is healthy-looking, but with five small blue-black marks. Organs normal; passed several worms after thymol treatment. Marks faded after thymol.

FIG. 2.—Male, aged 20, shows no signs of disease. Organs normal; tongue healthy looking, three small marks blue black; passed worms after thymol.

FIG. 3.—Male, aged 34, was in a very weak anæmic state; face puffy; legs swollen; marked jaundice; urine no albumen; heart dilated, complains of palpitation; tongue dirty yellowish clay colour, with blue-black edge; treated with thymol; passed large quantity of worms. Very much improved. Tongue changing colour.

[¹ We found it impossible to represent these colours accurately, so we have been content to indicate the situations of the markings merely, see illustrations.—EDITOR.]

FIG. 4.—Male, aged 20. No anæmia; organs normal; tongue pinkish, not quite normal colour; taken into hospital on account of tongue; has one large and two small blue-black marks; passed worms after thymol. Very much improved; marks fading.

FIG. 5.—Male, aged 29. Slight anæmia; tongue a little pale, with two blue-black marks; passed worms after thymol. Marks fading.

FIG. 6.—Male, aged 20. Slight anæmia; organs normal; tongue a little pale, with three blue-black marks; passed several worms after thymol. Very much improved; marks fading quickly.

FIG. 7.—Male, aged 29. A thin man, but no anæmia; organs normal; tongue normal in colour, with three very large marks, blue-black in colour and the edges fading away into red of tongue. Has no œdema; urine normal; passed large quantity of worms. Much improved, but marks are fading very slowly.

FIG. 8.—Male, aged 19. Not anæmic; organs normal; tongue healthy looking, with five small blue-black marks on tip; passed ankylostomes after thymol. Discharged cured, with marks quite faded.

FIG. 9.—Male, aged 19. Marked anæmia; no œdema; colour of face faded in patches; tongue pale, and has a very large blue-black mark on one side, and a smaller one on the other; passed ankylostomes after thymol. Very much improved.

FIG. 10.—Male, aged 25. Thin, wretched-looking man; well-marked anæmia; palpitation; no œdema; urine normal; has lost colour in face; tongue yellowish pink, the marks in this case are brownish with darker brown minute spots, like ground coffee; has passed ankylostomes three times after repeated doses of thymol. Very much improved; spots less brown.

FIG. 11.—Male, aged 25. Anæmic; organs normal; no œdema; tongue light pink, one large and two small marks, but of a washed-out black colour (not blue-black); passed ankylostomes after thymol. Improved.

FIG. 12.—Male, aged 30. Very weak and anæmic; lost colour badly about face, and is puffy; œdema of feet; tongue very pale, four small brown marks (nearly same as fig. 10); passed large quantity of ankylostomes after thymol.

FIG. 13.—Female, aged 22. Thin and anæmic; lost colour in patches on face; organs normal; tongue pale, both sides marked blue-black, some parts darker than others and edges run into red of tongue; passed ankylostomes three times. A little improved.

FIG. 14.—Female, aged 30. Very thin, anæmic woman; lost colour about face; tongue pale, with two very large blue-black marks; passed ankylostomes several times after repeated thymol. Slow improvement.

FIG. 15.—Female, aged 25. Very anæmic; face puffy; palpitation; no albumen; no œdema of legs; tongue yellowish pink, with many very fine brown-red spots at tip; passed ankylostomes twice after thymol. Spots fading.

FIG. 16.—Female, aged 24. Not anæmic; healthy looking; tongue bright pink, there is one large coffee-coloured mark, with small dark red-brown spots about it at side of tongue and a small one in middle; passed ankylostomes. Slow improvement.

FIG. 17.—Male, aged 25. Thin, anæmic condition; lost colour about face; palpitation; no œdema; tongue pale, one small blue-black mark; passed few ankylostomes. Nearly well; spot fading; general condition much improved.

FIG. 18.—Male, aged 21. Weak, thin, and very anæmic; palpitation; no œdema; tongue yellow-pink; three large marks, blue-black, but darker in some places than others; passed large number of ankylostomes. Very great improvement.

QUININE IDIOSYNCRASY LEADING TO HÆMOGLOBINURIA.

By AUBREY HODGES, M.D.Lond., M.R.C.S., L.R.C.P.
Medical Officer Uganda Protectorate.

THE following case would seem to be pretty clearly one of quinine idiosyncrasy leading to hæmoglobinuria, and a short account of it may therefore prove of interest to your readers.

Mr. S., a German, came under observation on November 10th, 1899, suffering from malaria. He had been two years in tropical Africa, and had had a good deal of "fever." Having heard much of Dr. Koch's theory of the relation of quinine to blackwater fever, and having also been told by his German doctor that quinine produced blackwater fever, he had sedulously abstained from taking the drug. But on one occasion when he was very bad with malaria, about a year ago, he had taken half a gramme of the sulphate, a few hours after which he passed black urine. The next urine he passed was clear, and in two or three days he was quite well. He had now had daily attacks of fever for several weeks.

When seen he was extremely anæmic, with waxy pallor, and the spleen was considerably enlarged, but what troubled him most was the persistent vomiting, so that he could not retain nourishment, and was become very thin and weak.

He had been in the habit of taking methyl-blue as a substitute for quinine, of which he had so great a horror that he could not be persuaded to try it. He was ordered nutrient enemata and was put on full doses of methyl-blue for two days, and then Warburg's tincture was tried. On November 15th, as there was no improvement, after consultation with his German colleague half a gramme of quinine sulphate was mixed with his morning enema without his knowledge. About mid-day he passed half a pint of port-coloured urine with a trace of albumen, but there was no alteration in his general symptoms, and the next urine passed, after five and a half hours, was normal. I determined to continue the quinine, and gave half a gramme daily for two more days, not only without recurrence of hæmoglobinuria but with distinct benefit to the general condition of the patient. I then gave half a gramme twice daily, still in enema, till at the end of the week he was much better, vomiting had ceased, and he was able to take food by the mouth and enjoy it. He was then told that he had been taking quinine, and readily agreed to take half a gramme twice daily by mouth for a week. At the end of that time he was able to start on a long

journey, taking a supply of quinine with him and having directions to continue taking half a gramme daily for at least three weeks.

He returned on January 15th, 1900, in good health, but shortly afterwards he had two sharp attacks of malaria, during which he was freely dosed with quinine without any recurrence of hæmoglobinuria.

The plasmodia found in this case are described as small discoid and ring forms. No pigment was seen and no crescents found.

THE SLEEPING DISEASE (DOENÇA DA SOMNO).

From the Portuguese.

REPORT SENT TO THE PORTUGUESE MINISTER OF MARINE BY THE SCIENTIFIC COMMITTEE SENT TO STUDY THE SLEEPING SICKNESS IN WEST AFRICA, ON FEBRUARY 21st, 1901.

(Continued from page 172.)

PART III. (condensed).

THE microscopic investigations were made on specimens taken from various parts of the nervous system. Some of the specimens were placed in absolute alcohol, some in Müller's fluid; sometimes they were treated by other methods, including impregnation by paraffin or cellordina.

The diplo-estreptococcus was found to exist in notable quantities in the vessels of the pia mater, in the lymphatic sheaths of the vessels of the cerebral and medullary capillaries. The glands at the bifurcation of the trachea exhibited numerous diplo-estreptococci. The preparations made by the method of Weigert did not show any nervous fibres in a state of decay or degeneration.

BACTERIOLOGY.

The only parasite constantly found in our investigations, made during life and *post mortem*, was the diplo-estreptococcus; the principal characteristics of this micro-organism we give below.

The preparations were made from the liquid obtained from lumbar punctures taken during life or at the autopsy. The micro-organisms were found disposed in pairs, more or less round in shape, sometimes slightly elliptical. The adjacent surfaces of each were slightly flattened, rendering the cocci somewhat hemispherical, so that they presented an aspect analogous to the cocci of Neiss, and to the diplococcus intracellularis meningitidis of Weichselbaum; usually there was a clear aureola round them, slight, but nevertheless quite perceptible. These diplococcal forms were not usually observed in the fluid drawn off during life; but in fluid examined from bodies ten or twelve hours after death it was observed that many of the diplococci formed themselves in chains of two, four, eight, or more.

In the majority of cases the micro-organisms were free to move in the secretion. Rarely do any occupy the protoplasm or nucleus of the cellular elements. The diplococci are, as a rule, more frequently found in the cerebral secretion than in the spinal fluid; although in some cases they existed equally in both.

They are easily stained by various aniline dyes, for example, by the alkaline blue of Loeffler, or the phenicated thionine of Nicholle. Treated by Gram's method (we stain them generally by a modification of the Nicholle system), their behaviour is irregular, some taking the colour, others not, and some to an intermediate extent, so much so that in the same chain of diplococci the cocci may be stained of diverse tints. After one or two experiments on the cultures I made—especially on the solid ones—I obtained uniform results by the Gram-Nicholle method. The diplo-estreptococcus generally cultivates very badly in all the ordinary media. In the meat extract, or "agar," or mushroom, simple, or with glycerine, they generally fail every time. The liquid proposed by Martin for the culture of the bacillus of Loeffler, and obtained by macerating the stomach of the pig, gives very much better results, especially if we make it solid by the addition of "gelose." The best solutions are those containing ascitic fluid, for example, a mixture of this liquid with the extract of meat in equal parts; or the medium recommended by Kiefer for the cultivation of the gonococcus, substituting the extract of beef for the macerated solution of the pigs' stomach, or mushroom.

In the liquid medium containing the peritoneal secretion there was noticed in eighteen to twenty-four hours, at a temperature of 35° to 37° C., the appearance of growths not very abundant, but manifesting themselves by a slight turbidity, generally of a uniform nature. This turbidity increases consecutively, but without attaining a great degree. At the end of four or five days there is a small sediment of the bacteria visible in the bottom of the glass, and the liquid becomes very much more limpid. In Martin's cultivation medium we saw the same phenomena, but in a greater degree. In "solid" extracts, to which I have referred above, there began, at the end of twenty-four hours and at the same temperature, to be visible, small, round, or oval colonies of a greyish white colour, very transparent and with a somewhat brilliant surface. Their growth at the end of the third or fourth day never exceeded 0.2 mm. to 0.3 mm. Observed under the microscope with a very low power they present themselves as composed of very small granulations, possessing folds but not clearly marked. Later on it is easy to distinguish two zones, one central, of a darker colour, the other at the periphery. When the crop is abundant we see them join together in a partial manner at certain points. With gelatine we never succeeded in obtaining cultures. The preparations made from cultures from various morbid products varied a little, according as they were treated as solids or liquids. In the solid medium numerous chain-like forms appear by the side of the micro-organisms disposed in pairs and isolated one from another. The chain-like forms are always easily recognised, and represent the diplococci. The dimensions of the diplococci vary between 0.7 μ to 0.8 μ , but including the aureola, they attain the size of 2.5 μ to 2.7 μ . They are always in a quiescent state.

The diplo-estreptococcus, which we have described above, had never, so far as our investigations go, any great pathogenic power on animals. The "cobaias," injected in the peritoneal cavity, and the pigeons

in the pectoral muscles, resisted large doses of recent cultures. Rabbits and mice were, however, quite sensible of the effects: they were sometimes killed outright.

With 1 cc. to 2 cc. of the culture, of twenty-four or forty-eight hours' growth in liquid medium with peritoneal secretion, death supervenes in a period of from two to seven days; the micro-organisms were found in greater or lesser number in the blood and in the spleen. In the mouse the doses of 0.5 cc. to 1 cc. of the culture of meningeal secretion, for example, inoculated under the skin, caused death by septic poisoning at the end of two to four days.

The spleen of the animals which succumbed to this experimental injection remains at the usual size and of the general consistence. There were no morbid microscopic alterations visible, save the renal congestion in the rabbits which resisted the disease for a longer period.

The observations have been made on the liquid obtained from punctures in the lumbar region, on lymphatic glands removed during life or during the autopsy, in the blood taken by an aspirating syringe and sterilised, and in the meningeal fluid from dead bodies. The lumbar puncture was done nine times. In six patients it gave positive results, the bacteria being easily isolated; in three the examination of the direct preparations and the cultures gave negative results.

Our experience has taught us that the fluid from lumbar puncture is the best medium of culture, adding an equal quantity of Martin's extract of double strength, and placing the whole at a temperature of 37° C.

Three times glands were removed during life, from two of which positive results were obtained, and a negative result with the third. We made investigations of a bacteriological nature of the blood of four patients. The blood was extracted by a Debove's syringe sterilised at 120°, and the skin was carefully disinfected. We found abundant growths in the cultures—liquids as well as solids—immediately after the growths appeared and before it had time to separate by means of coagulation.

In the meningeal fluid from dead bodies the diplo-streptococci were found in a pure state in thirteen cases in which we made autopsies. We termed the microbe we isolated the diplo-streptococcus. The first part of the name is justifiable, as it presents itself under the form of a diplococcus with great regularity and frequency. The second termination is justified by the manner in which it conducts itself in the preparations; but we cannot say just now whether or not the micro-organism we describe is or is not a transition type between the microbe of Traenkel and the estreptococcus.

The result of our investigations has led us to the conclusions, that: (1) Sleeping sickness is a meningo-encephalic affection; and (2) that it is due to a microbe which we term the diplo-streptococcus.

THE DURATION OF THE LATENCY OF MALARIA AFTER PRIMARY INFECTION. AS PROVED BY TERTIAN OR QUARTAN PERIODICITY, OR DEMONSTRATION OF THE PARASITE IN THE BLOOD.

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(Translated from the Italian by St. Clair Thomson, M.D.Lond., F.R.C.S.)

(Continued from page 176.)

PART III.

ATTACKS AT LONG INTERVALS.

We have seen that with regard to shortly recurring attacks, it may be generally stated that the period of latency in all three forms of malaria is the same as that of incubation. Let us now proceed to examine the period of latency in attacks recurring at long intervals.

Manson refers to the case of his son who was inoculated with spring tertian in 1900, and had an attack in 1901 after full nine months' complete apyrexia, and this in spite of three months' continual quinine treatment after the cessation of the paroxysms, and without his ever having left London, a notoriously non-malarious place.

That attacks recurring at long intervals may come on after a very long period of apyrexia has long been recognised; and this case of Manson, well-defined as to the nature of the attack, is but a repetition of what has frequently been observed in malarial districts. Its importance, however, is much greater because the same febrile tertian cycle was repeated, and the microscopic examination of the blood revealed the presence of endoglobular parasites of spring tertian in a person to whom the infection had been communicated experimentally, and who had always resided in a non-malarious district, which excludes the possibility of reinfection. It is also clear that in such a case a continued quinine treatment is of no avail, since it did not completely cure the patient, who persevered with it for three months after the disappearance of the paroxysms.

Such cases are not rare. I remember one similar, observed by Professor Bignami in the hospital of Saint Galla in Rome. A patient, after a wound, had an attack of typical quartan fever. This patient had been apyretic for many months, and then suffered an attack after a long interval of quartan infection experimentally provoked a long time before, and treated with quinine for a long while afterwards. Neither could reinfection be logically supposed in the case of this patient, seeing that his condition (hemiplegia) had detained him in the hospital for many months, and he had certainly not moved since he was inoculated with quartan.

It is not a rare case for a medical man to be summoned to the mountains to treat a patient suffering from an attack of malaria after many months of apyrexia, frequently following one of the commonly recognised determining causes. But these cases have not been well studied, and are only mentioned here

and there as among the curiosities of daily medical practice.

In coming to the results of my observations I must note before all:—

(1) That the period of latency of malarial fever in general may extend for many months.

(2) That an attack after a long interval does not occur except by the intervention of one of the recognised determining causes noted, and never spontaneously.

(3) That it occurs in spite of all treatment followed, whether temporarily or continued during the whole period of latency, by quinine, arsenic, iron, iodide of potassium, &c., accompanied by good, wholesome, nourishing food, and all the conditions of a healthy life advised at the time.

(4) That the attack is not influenced by age or sex.

With regard to the second assertion it may be affirmed that there is no fixed period of latency beyond which the attack cannot recur, on the contrary, every time a person infected with malaria is exposed to one of the recognised causes, the effect is to bring on a fresh access of fever. I have not found a case in which the limit of time exceeded thirteen months (in spring tertian), yet even when that limit is reached, it may be deduced from the cases observed, that the said period would have proved to be much longer if any of the recognised causes of relapse had intervened later.

To come to facts. It is not rare to find persons who, having caught the tertian infection and recovered from the first paroxysm without therapeutic intervention, have returned to districts free from malaria, where some have continued the quinine treatment they had commenced for two or three months. Several months later, returning to a malarial district, these persons, after a few days or hours, have been seized with attacks of a tertian type, though frequently they have continued or recommenced the quinine treatment. Should these be regarded as cases of recurrence?

I think that the majority of such cases should be so regarded, absolutely so in cases where the fever has reappeared after a few hours' residence in a malarious district. It is true that many facts raise a doubt as to whether it may not sometimes be possible to contract true malarial infection in a district previously considered non-malarial,¹ but it would be senseless to admit the theory of fresh infection, especially as in every case the patient suffered from the same form of malaria as before.

It is not difficult to observe these cases in isolated individuals, or in the families of labourers, although many malarial patients in July and August return to their own country as soon as they have left the hospital, and only come down in October, November and December, to attend to the ploughing and sowing of the fields.

Chapter 1.—Spring Tertian.

In 152 cases of spring tertian studied by me, though during first infection, I observed that the attack recurred within an interval varying from three to four months. In all the 152 cases I can certify that they had not returned to a malarious district in the meanwhile, nor suffered from attacks of fever. I observed that in all these cases the attack came on after the intervention of one of the recognised determining causes. Here follows the principal data respecting the period of latency in these 152 cases.

The attack recurred:—

(A) Within two months from the beginning of apyrexia in 60 cases, and these patients were nearly all overtaken by it while undergoing the quinine treatment.

(B) Within seventy days in 29 cases.

(C) In ninety days in 20 cases.

(D) From the 90th to the 120th day of apyrexia in the remaining cases.

None of these patients resided in a malarious district after the first apyrexia; in spite of this some of them had afterwards two, three, and more attacks at short intervals; in 102 cases the attack occurred while the patients were undergoing the quinine treatment, which was combined in almost every case with the iron-arsenic treatment.

The following Table clearly shows the results ascertained in the cases of 14 patients who had never taken quinine.

| Number of Cases, Age, Profession | Duration of Apyrexia before attack | Residence during Apyrexia Height | Duration of Apyrexia on Return to Malarious District | Cause of Attack | Duration of Apyrexia after Intervention of Cause of Attack | Season of Attack |
|----------------------------------|------------------------------------|----------------------------------|--|------------------|--|------------------|
| (1) 1 man, 26 | 50 days | Aquila, 721 m. | 29 hours | Rain | 9 hours | Oct. |
| (3) 1 man, 40 | 58 days | Pausola, 500 m. | 38 hours | Rain | 22 hours | Oct. |
| 1 woman, 39 | 60 days | " | " | Cold | " | " |
| 1 child, 10 | 58 days | " | " | Under-feeding | " | " |
| (6) 1 man, 50 | 69 days | Monte Tancia, 800 m. | 45 hours | Rain | About 18 hours | Oct. |
| 1 woman, 47 | 73 days | " | " | Damp | " | " |
| 1 youth, 20 | 68 days | " | " | " | " | " |
| 2 men, 18-19 | 90 days | " | " | " | " | " |
| 1 boy, 12 | 88 days | " | " | " | 12 hours | " |
| (3) 3 men, 26-30 | 90 days | Preturo, 792 m. | 26 hours | Cold | 16 hours | Dec. |
| (1) 1 boy, 16 | 92 days | Norcia, 603 m. | 30 hours | Wound in abdomen | 10 hours | Dec. |

¹ This is known with regard to certain districts; further, in long years of observation I have gathered copious materials from individuals from a mountainous district hitherto reputed non-malarious, who had never been ill and yet were infected with malaria when they left the said district (see "Some Observations on the Epidemiology of Malaria," by Dr. Attilio Caccini. --*Polislinico*, 1901-1902).

I further add several very convincing cases which prove that in patients not treated with quinine, in whom the primary infection of spring tertian has spontaneously exhausted itself, the attack recurring after a long interval (within two and twelve months of apyrexia), always comes on after the intervention of

one of the recognised determining causes aforesaid. These cases are specially important, as in dealing with educated and intelligent persons the facts gathered are more trustworthy.

The case in point is that of a whole family of ten in easy circumstances, six of whom while at Nettuno contracted the infection of spring tertian in July, 1900.

(A) P. G., 50, employee.

(B) P. L., 40, mother of three children.

(1) P. M., 25, employee.

(2) P. R., 20, officer in the army.

(3) P. M., a girl of 17.

(C) F. E., maternal aunt, 51.

I was called in to this family by the doctor who attended them, when I made an examination of the blood and collected the information given there. None of them had ever had malaria; after a residence of from fifteen to twenty days at Nettuno all six were seized with fever of a clearly defined tertian type. They removed from Nettuno to a locality in the Alps where they had property, and the attacks ceased spontaneously. F. E. alone (see C) having several attacks at short intervals, stopped them with quinine taken rather irregularly for nearly two months. They remained on their estate for three months and a half and P. R. (see No. 2) was then obliged to join manoeuvres in the field. On his first night in camp, after a long ride, and wet with perspiration, he was obliged to expose himself to the sudden cold of the evening. Within twenty-four hours the attacks of tertian fever commenced and he stopped them with quinine. When the attacks came on it was hardly forty-eight hours since he left his Alpine residence.

The rest of the family returned to Rome towards the middle of November; none of them had suffered from the fever for four months. Towards the end of November P. L. (see No. B) had a fresh attack of tertian fever following a miscarriage, the fever appearing twenty-six hours after the miscarriage. An examination of the blood showed the presence of the endoglobular parasite of spring tertian.

No. A (P. G.) took part in a swimming race towards the beginning of December and was immersed in cold water for some minutes; thirty-two hours later he had an attack of tertian with the positive presence in the blood of the plasmodium of spring tertian.

No. C had a relapse in December after an attack of cardiac asthma, to which she was subject.

Of the members of this family who had contracted malaria at Nettuno, that is, six out of ten, No. A and No. 3 remained almost immune. In order to counteract the malarial infection and in the hope of avoiding fresh attacks, the six patients entered upon a severe quinine treatment (1 to 2 grammes a day), which they continued until March 1st, 1901. In July, 1901, the family went to Viareggio; they had only been there three days when every one of the six had an attack—a few hours before they had been swimming and bathing in the sea. It is also to be noted that in the meanwhile (from December, 1900, to July, 1901), they had not moved from central Rome.

Thus the first relapse occurred after about two and a half months of apyrexia in one of these cases, and after four months in three of the others, that is, in

four out of six. In every case the attack followed upon some debilitating cause.

The possibility of reinfection in a mountainous district may be excluded, as only those members of the family were attacked who had contracted the infection at Nettuno, while those members (four) who had not taken the malarial infection at Nettuno or any other place, never suffered from fever, though leading the same kind of life as the other members of the family. The quinine treatment had no effect whatever.

The attack recurring after a long interval exactly resembles true first infection, that is, the paroxysms of fever may be more or less regular; attacks may or may not occur at long or short intervals and it reacts equally against the quinine treatment. But whereas regular systematic treatment prevents attacks recurring at short intervals, it does not prevent those recurring at long intervals, which come on after a space of time which may reach twelve months of apyrexia. The attack always occurs upon the intervention of any of the organically debilitating causes mentioned. Thus with patients treated with quinine and divided into categories according to the method of the treatment, every category shows the same percentage of attacks recurring at long intervals. In every case the attack followed upon the intervention of one of the debilitating causes noted. Any patient guarding against debilitating accidents and observing a regular diet, may remain free from attacks of fever for a long while (six to seven months), but suffers a relapse after that time upon exposure to cold, fatigue, wounds, or illness.

One of the longest examples of latency which I had the best opportunity of studying was the following: Between September 10th and 16th, 1901, thirty-eight Calabrians by birth, presented themselves at the Hospital Santo Spirito, all suffering from spring tertian; their ages ranged from 7 to 60 years; all were of a robust constitution and stokers by profession. They all came from the same place (Valle d'Inferno), a place still noted as very malarious. I was surprised at the identity of the place from which they came, their birthplace and calling; all the patients gave the same account.

They had reached Rome and established themselves at Valle d'Inferno (where most of them were employed at the Bolognese furnace) only forty-eight hours before. While they were resting, being in a strong perspiration from their labour at the furnace, the whole band (a hundred Calabrians from the same place) were caught in a sudden cold and heavy shower. Immediately after an epidemic of spring tertian broke out among them. They all asserted that the fever broke out within from ten to twenty-four hours after the rain, and barely forty out of a hundred escaped it. The sixty who fell ill had already suffered from tertian fever in the summer of 1900. I then repaired to the spot and collected the following facts.

In July, 1900, a large band of Calabrian mountaineers had contracted spring tertian at Foggin. They returned to the mountains in August and all the patients followed the quinine treatment until the end of December, and with the exception of some who had attacks in August and December, in the majority apyrexia had lasted from the end of August. On September 8th, 1901, that is, fourteen months after

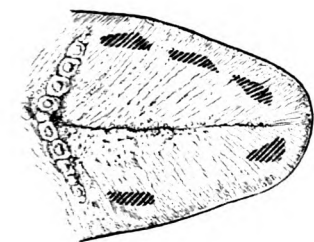


Fig. 1.

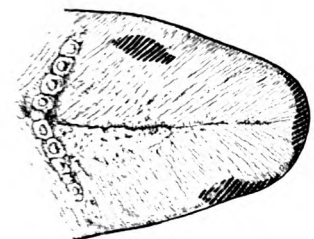


Fig. 2.

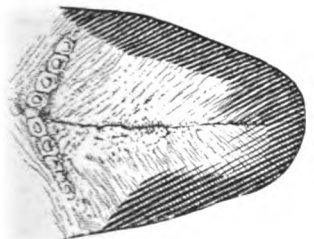


Fig. 3.

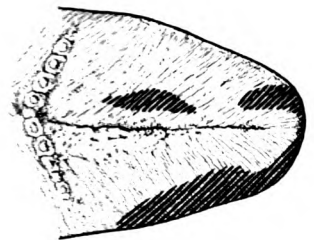


Fig. 4.

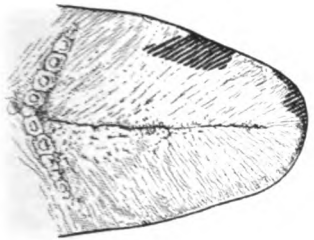


Fig. 5.

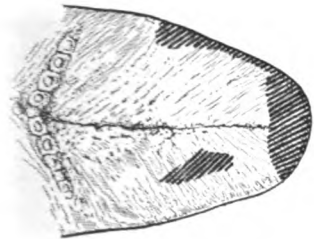


Fig. 6.

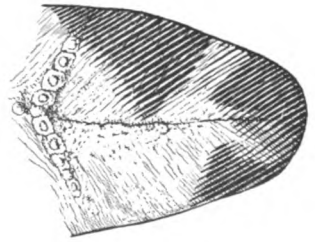


Fig. 7.

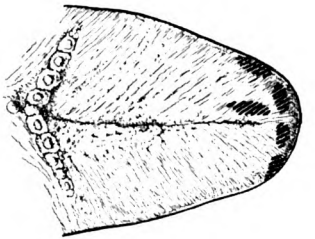


Fig. 8.

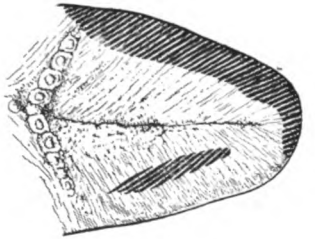


Fig. 9.

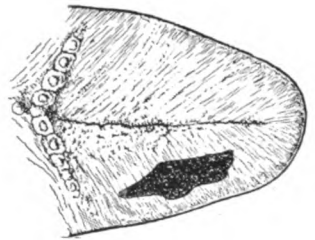


Fig. 10.

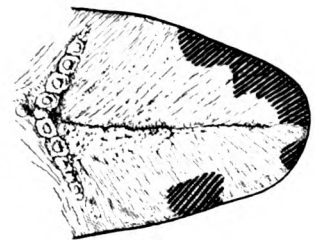


Fig. 11.

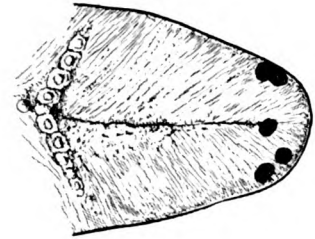


Fig. 12.

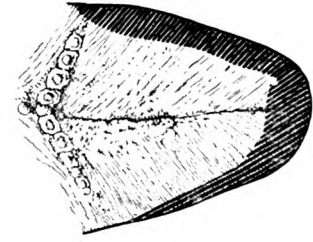


Fig. 13.

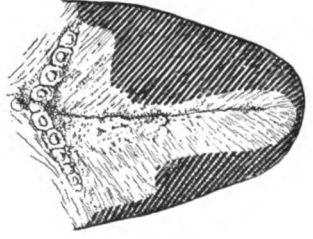


Fig. 14.

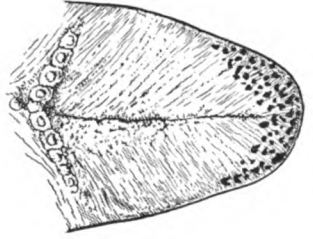


Fig. 15.

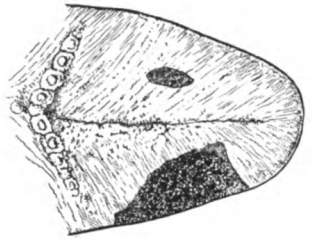


Fig. 16.

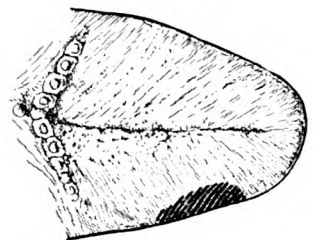


Fig. 17.

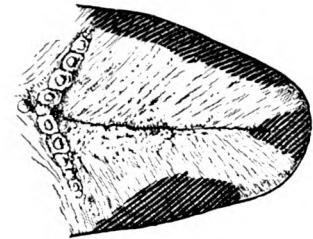


Fig. 18.

To illustrate peculiar Marking of the Tongue in Ankylostomiasis. By PERCY H. DELANEY, L.R.C.P., L.R.C.S.I.,
Government Medical Officer, Leguan, British Guiana.

primary infection, a band of 100 Calabrian labourers were engaged, including 75 of the aforesaid band. They arrived in Rome by rail after a journey of twenty-four hours, on the morning of September 9th. That evening the stokers were divided into two groups.

(A) Eighty-one who were exposed (including 62 of the aforesaid band).

(B) Nineteen who were under shelter (including the remaining 13 of the said band who had suffered from malaria the year before).

After the rain the attack recurred exactly in the case of the 62 who were exposed to it. The first cases appeared in the morning of the 10th and increased rapidly on the following days. In the 62 cases apyrexia had lasted:—

(A) In 20 for thirteen months.

(B) In 15 for ten months.

(C) In 10 for nine months.

(D) In 12 for eight months.

(E) In 3 for seven and a half months.

(F) In 2 for seven months.

All had continued the quinine treatment for three or four months at a dose of 0.50—1.50 grammes a day, or thereabouts.

This is the most conclusive example I have seen of long interval in spring tertian, apart from the case of private practice already quoted.

The period between January and June is specially well adapted for the study of attacks at long intervals when recurrent attacks of all kinds of malaria are most frequent, while the first attacks are unknown, or at least very rare, as proved by the authors,¹ and, according to what I have myself observed in four long years devoted to the study of the malaria epidemic among the patients of the hospitals in Rome. Thus I have frequently observed the recurrence of the attack from January to June in persons apyretic from the end of December who had certainly not since resided in a malarious district. In every case it was easy to trace the cause of the relapse.

Chapter 2.—Quartan.

I have already mentioned a case referred to by Professor Bisagni in which an attack of quartan after a long interval followed upon a fall many months after the first attack of quartan brought on by inoculation.

Other cases were somewhat rare (six in all) in which I was able to study the duration of the period of latency in attacks recurring after many months of apyrexia. However, in all six cases the attack followed a wound, rain, or a chill, and in one case an attack of clearly-defined quartan followed upon recovery from typhoid, and an examination of the blood demonstrated the presence of endoglobular quartan parasites.

In all six cases the attack recurred after six to ten months of apyrexia. But cases of a shorter period of latency were very frequent (one of three months), and calling to mind what I have said previously, and that in all and every case the evidence of some determining cause preceding the attack was clear, it may

be judged how rare are the cases of very long latency, for it is difficult for a quartan patient to escape some determining cause for more than two or three months. It is well to remember that in quartan also the attack recurs in spite of quinine, iron and arsenic treatment.

QUARTAN PATIENTS RELAPSING AFTER A LONG INTERVAL.

| Description | Date of Infection | No. of Relapses at short intervals | Residence and Duration of Apyrexia | Cause of Relapse | Apyrexia after Cause | Date of Relapse |
|---------------|------------------------------|------------------------------------|------------------------------------|-------------------------------------|----------------------|-----------------|
| Ploughman, 40 | October, quinine 3 months | 3 relapses till December | Sondrio, from December to August | Rain | 70 hrs. | 2 Aug., 1900 |
| Ploughman, 36 | June, 1898, quinine 3 months | Relapse, October, 1899 | Alps, July to August | Chloroform | 39 hrs. | 6 Aug., 1900 |
| Contadina, 24 | October, 1899 | 2 relapses November | Sondrio, April, 8 months | Fall from a cart, pregnant 6 months | 55 hrs. | 6 Aug., 1900 |
| Contadina, 16 | August, 1900 | April from December, 2 relapses | Central Rome | Enteric | 5th day | 6 Sept. 1900 |
| Man, 19 | August, 1900 | April, 8 months, | Central Rome | Bath in Tevere | 66 hrs. | 7 July, 1901 |
| Ploughman, 32 | September, 1900 | 4 relapses | Pausola, 3 relapses | Excessive fatigue | 50 hrs. | 22 Sept. 1900 |

Chapter 3.—Fevers: Summer—Autumnal.

Is there a recurrent attack at long intervals in summer fever?

In all the number of cases studied, I have observed a true attack recurring after a long interval in four patients only. In two of these cases the attack occurred after sixty days of apyrexia and complete health; in the third case after ninety days, in the fourth after eight full months.

In the four cases the attack came on in the first after a wound, in the second after heavy rain and many hours' exposure to the north wind (tramontana); in both cases apyrexia had lasted sixty days. In the third the attack followed upon a surgical operation (extirpation of inflamed inguinal lymph glands under chloroform) after ninety days of apyrexia; in the fourth the attack came on after bathing in the river, when the patient narrowly escaped drowning, and in this case the fever appeared only twenty-four hours after the accident.

It is well to note, however, that the other three cases belonged to the group of malignant tertians, and in the last case the summer fever was of the quotidian type.

All these observations of mine agree with many facts recently proved, that is:—

That the summer and autumnal fevers are the worst and most dangerous in their symptoms and pernicious

¹ "Papers of the Society for the Study of Malaria." On the annual course of the malaria epidemic.

nature; that they easily recur at short intervals in spite of quinine treatment; they are, however, more easy to be definitely overcome. In fact, though it is very common for patients in summer fever to suffer many recurring attacks in a short space of time after primary infection, it is very rare to find the attacks recurring in spring, and absolutely exceptional to find the summer form after April. If it is possible that in some cases the recurring attacks pass this limit of time it is at least extremely rare to find any manifestation of fever in April, May, and June, and when these occur they are short, non-typical and abortive. I have never found any case of pernicious fever after February, and it is worthy of note that in the hospital of S. Spirito no dead body escapes *post-mortem* examination when there is the faintest suspicion that malarial infection, active or latent, may have had a share in the death. I can affirm with absolute certainty that in 1899 the last serious case of malaria occurred at the beginning of February, the last autopsy took place about the middle of January. In 1900 the last case of pernicious fever was on February 1st, but there was no autopsy of a pernicious case after the middle of December. In 1901, after the grave epidemic of 1900, the last serious relapses occurred when February was well advanced. The last subject of pernicious fever examined was on January 15th.

At present the malaria season, both in S. Spirito and the Campagna, may be said to be over after the middle of November; no case of primary infection has occurred since the middle of October, though some cases of serious relapse with pernicious symptoms still occur; the last autopsy was on November 1st, 1901. This was in spite of preventive measures, for there is nothing to prevent a sudden increase of malaria and pernicious fever, as this year the epidemic has shown great increase in the number and gravity of the cases.

What I have said is opposed by the fact that it is not rare for a patient to continue all the winter and a good part of spring with the summer parasite in the blood; I have found this in four patients. But not one of them showed in the meantime a feverish temperature or other morbid phenomena which might have led to a suspicion of masked attacks of fever. In all four the examination of the blood yielded a negative result after the beginning of May, and none of them relapsed in the following season.

A fact noted by several observers is that while the new malarial season is imminent there are no cases, or scarcely any, of summer malaria with crescent bodies in the blood; for my part I have never been able to find crescent forms after April, and very few in that month. *In May and June, however, I have never found a crescent.*

This does not prevent an occasional case in the country, so Martirano assures us;¹ it is certain, however, that the above are the facts proved by observation among the patients of the hospital; the discrepancy is perhaps due to the fact that patients with crescents do not always have attacks of fever and

therefore do not come to the hospital, and therefore these have not the rest, good food, and treatment, enjoyed by the patients in the hospital, the action of which influences the disappearance of the "*gametes*" from the circulating blood; while the effect of the labours in the field, on the contrary, is to make them complete the circle, and perhaps provoke a fresh generation, and therefore Martirano found true and real relapses among the country people.

Perhaps, however, the attack will be short and light (in fact, no case of pernicious fever is cited in the spring), and the labourers, bound to the land from which they expect subsistence, will not have recourse to the hospital, even though suffering slightly from fever. It is therefore supposed that conditions of place have no influence, and that the results do not vary year by year; and it seems to me that this should be taken into consideration, since Dionisi, even at the height of the grave epidemic in 1899, could find only one case of crescents, and even these were very scarce, in the whole of Maccarese—truly a strange thing when one considers that upon this lack of crescents and infected *Anopheles* was to follow the serious malaria epidemic which was to break out in Rome in 1899.

But I do not wish to dwell much upon this here, as in another work to be shortly published, in which I treat of the epidemiology of malaria, and especially of the course of the epidemic as studied in the years 1898, 1900, and 1901, I have set forth these facts in sufficient detail.

All that I have said is simply intended to clearly show that summer fevers, grave in their symptoms and prognosis, are the least obstinate in relapses; and that it is very rare (at least if not exceptional) for relapses to occur after a long interval, especially when spring is well advanced. In contrast to this is the fact that in spring fevers, on the contrary, relapses at short intervals are more frequent, the symptoms less grave, and the cure more obstinate. This may perhaps be explained by the fact that in our climate relapses of slight tertian and quartan are very frequent in the spring (so much so that these complaints are justly called spring diseases), and these attacks, recurring until the beginning of the new season may, every time they occur, exactly resemble true cases of primary infection; whereas this is not the case with malignant tertians, which, at least in my opinion, exhaust themselves in winter and spring.

PART IV.

CONCLUSIONS.

1. Malarial infection admits of relapses at short or long intervals.

2. In attacks at short intervals the period of latency approximately equals the period of incubation, and therefore: (a) In spring tertian varies from one to eighteen days; (b) in malignant tertian it varies from five to eighteen days, but generally occurs between the fifth and ninth day; (c) in quartan the duration of the period of latency is undetermined, for the latency ceases upon the intervention of any of those conditions which we have seen to be determining causes of relapse.

¹ "Papers of the Society for the Study of Malaria," vol. ii., p. 249, *et seq.*

3. Quinine treatment influences the duration of the period of latency to the extent that, in patients who have followed it systematically, the latency reaches its maximum duration, and in these the number of attacks are fewer.

4. The quinine treatment has no influence to prevent attacks at long intervals.

5. Attacks at a long interval invariably presuppose the intervention of a determining factor.

6. In spring tertian the attack at a long interval has a period of latency varying, as far as I could ascertain, from three to thirteen months.

7. In quartan the attack at a long interval is not clearly differentiated from that at a short interval; both are influenced by the kind of relapse and the importance of the intervening accidental factor, therefore the latency does not cease until the appearance of the latter.

8. The attack at a long interval in summer malaria may be said to have a latency varying between two and eight months, always remembering that the maximum limit of eight months was only found by us in one case—that of a patient suffering from summer infection, with fever of the quotidian type.

9. The attack at a long interval is very frequent in spring tertian and rare in quartan (if only because of the difficulty with which the patient can escape some determining factor of relapse, seeing the special obstinacy of quartan infection and its peculiar liability to relapses).

10. Tertian, spring, and quartan infection are more tenacious and liable to relapses. The prognosis with regard to relapses is much more serious than in malignant tertian, though the symptoms of the latter are more serious and dangerous, the attacks recur readily at short intervals, but there is no relapse at a long interval, or it is most exceptional after two months' apyrexia.

THREE LECTURES ON BILHARZIA,

Delivered at Kasr-el-Ainy Hospital, Cairo.

By FRANK MILTON, M.R.C.S.

Surgeon to the Hospital.

(Continued from page 170.)

LECTURE II.

SYMPTOMS AND SIGNS OF BILHARZIA.

GENTLEMEN,—I have promised to-day to discuss the symptomatology and diagnosis of bilharzia, and I intend to speak of the symptoms peculiar to the various organs commonly attacked by the disease in the same order as I took them when discussing their pathology; for although the disease has a common origin, its symptoms are necessarily widely varied according to the site of its manifestations.

THE BLADDER.

To begin then with the bladder. The earliest symptom of bilharzial disease of the bladder in the great majority of cases is hæmaturia of a peculiar and characteristic kind. The amount of blood lost is as a rule insignificant, and would probably never be noticed by the patient if it were mixed with the urine as it was passed; but the peculiarity of this hæmorrhage is that the few drops of blood which are lost

are voided either mixed with the last few drops of urine, or else escape from the urethra after the act of micturition is altogether finished, whereby the attention of the patient is caught by the marked difference in the appearance of the last part of the evacuated fluid. In a large number of cases the hæmaturia begins without any subjective symptoms, but as a rule, soon after the patient notices the loss of blood he begins to complain of pricking or scalding in the urethra during micturition, together with a sense of pain or weight in the perineum. In a few cases even before hæmaturia is noticed the patient complains of pains above the pubes, which may even extend up into the lumbar region, but this is rare and probably only occurs in those exceptional cases in which the eggs are first deposited in the muscular coats of the bladder instead of, as is usually the case, in the mucous and sub-mucous coats. The amount of blood passed is seldom excessive, even in well-established cases, but it may at times be increased by accidental causes, such as excess in food or drink or over-exertion, but this increase lasts only for a short time and ceases with the removal of the cause. Directly depending on the hæmorrhage, however, is another condition which may give rise to most serious symptoms, and that is when hæmorrhage takes place in the bladder to such an extent that the blood cannot be expelled and a solid clot is formed in the cavity. The condition which then arises is most serious, for not only does the bladder become distended with clot, but the urine coming down from the kidneys cannot be expelled, and a condition of acute retention is superadded, and the patient's condition is an extremely grave one. The symptoms are those of retention of urine with great and rapidly increasing distention of the bladder, and unless relieved, and that speedily, the patient passes into a state akin to uræmia and dies.

The frequency of micturition is not increased as a rule until the disease has lasted for some considerable time and a condition of irritability has been set up in the bladder, or until secondary changes have occurred and the symptoms of cystitis are added to those due directly to bilharzia. A very large number of patients infected with bilharzia suffer for a considerable time from the characteristic hæmaturia without any other symptom than a certain amount of uneasiness, or it may be even scalding, during micturition, due probably to the different specific gravity of the last few drops passed, and recover and never have any further trouble. Indeed this uncomplicated bilharzia is so common that very many sufferers from it never come under treatment at all, looking upon it as a thing which has to occur in the ordinary course of affairs, and therefore not worth troubling about. The cure in these cases is probably due to the death of the worms and the gradual complete throwing off of the eggs by the bladder. In this connection the question naturally arises as to the longevity of the parent worms, and unfortunately this is one of the many things still unknown in connection with this disease. It is probable that the life of the bilharzia is a long one; those cases which undergo a spontaneous cure are probably cases where a few embryos have been taken in by the host on

some particular occasion and the infection has never been repeated, and in these cases, although the statement of patients, even of the better class, who have recovered are generally very vague, yet most of them agree in putting the period of time during which they had hæmaturia at about two years. Sonsino's case before referred to would put the possible longevity and sexual activity of the parasite at nine years.

HÆMATURIA.

The immediate cause of the peculiar form of hæmaturia occurring in this disease would appear to be that the mucous membrane of the bladder being invaded by the eggs is in a state of irritation, and to a certain extent of hyperæmia, the presence of the egg-infarcts also must to a certain extent interfere with its normal resilience and elasticity, thus when the bladder has emptied itself of its contained urine, and has contracted down to its smallest limits, the hyperæmic, thickened, and comparatively inelastic mucous membrane is crushed on itself, some of the superficial vessels are ruptured, and the blood thus extravasated is forced out after the urine. The urine itself in this stage of the disease is as a rule normal in colour and reaction, and if the portion first passed is caught in a separate vessel nothing abnormal is as a rule found in it. If, however, the whole of the urine is collected together and allowed to stand in a conical vessel, as it settles minute flakes and flocculi will gradually be seen to accumulate at the bottom, which on examination under the microscope will be found to consist of red blood corpuscles, leucocytes, epithelial cells and bilharzia eggs entangled in mucus. In a certain number of cases the hæmaturia after having continued for a time instead of gradually becoming less and finally ceasing, continues and tends to increase, either owing to the presence of an unusually large number of worms being originally present in the victim or to fresh infection, and presently symptoms due to alteration in the bladder, with impairment of its functions, are set up. At first the symptoms resemble those due to vesical catarrh of a not very acute form, thus there is a certain increase in frequency of micturition, an undefined sense of not having completely emptied the bladder after the act, with scalding during the whole time of passage of the urine, with perhaps some little hesitation in beginning. At this time, and as the symptoms would indicate, no irreparable damage has been done to the bladder, but after a further period, which may be shorter or longer, according to the extent and rapidity of the deposition of the eggs, changes are set up in the mucous membrane of the bladder which are incurable, the changes being due to the definite formation of the thickened patches and the commencing organisation of their fibrous tissue. When the mucous membrane has become so far affected that these raised, hardened patches are developed on the one hand and ulcerative fissures are formed in them by necrosis on the other, the symptoms will pass from the comparatively slight ones of vesical catarrh to the more severe ones of cystitis, and often after but a short interval to those of septic cystitis. The septic germs have either been introduced from without by the passage of instruments or else by micro-organisms finding their way

into the bladder up the urethra, whose canal is kept open by the continuous dribbling of small quantities of urine; for when once the bilharzial patches are well advanced the complete contraction of the bladder is prevented, when there will always be a certain amount of urine remaining unexpelled, which keeps dribbling away almost constantly. When once sepsis is developed in the diseased bladder all hope of curing the patient is practically over, for owing to the nature of the affection, with deep ulcers running in all directions into the overgrown and unhealthy mucous membrane, and the presence of a continually renewed supply of putrescible matter, no amount of cleaning with antiseptics, either by means of washing out the cavity or by medication through the blood, can ever exterminate the septic germs when once they have got a hold. The bladder, too, has probably by this time lost most of its functions, that is to say, it can no longer either retain the urine brought to it from the kidneys nor expel it properly by the urethra, and it becomes a mere diverticulum met with by the urine on its passage, whose contents are a foul mass of blood, sloughs, bilharzia eggs and decomposing urine. The inability of the bladder to expand causes it to endeavour to pass on the urine as it enters; the bladder is thus kept in a condition of continual spasm or tenesmus. The patient has now reached the most distressing stage in this ghastly disease; the necessity of trying to empty his bladder is incessant day and night, the scalding due to the passage of the foul, irritating urine along the urethra is both constant and severe, tenesmus is almost continuous owing to spasms of the irritated ejaculatory muscles, there is constant pain in the perineum and above the pubes from implication of the prostate and the bladder walls, and a constant liability to acute retention of urine owing to blockage of the urethral by a slough or calculus. When to all this is added the complication of pyonephrosis from extension of the septic conditions backwards along the ureter, the patient's condition is hopeless indeed. The development of carcinoma, which may take place at this stage, does not add greatly to the patient's sufferings, the only symptom it is likely to give rise to being an increase of the hæmorrhage; but this is only important in that it may hasten the termination of a case which can only end in death.

(To be continued.)

BUBONIC PLAGUE.—Joseph J. Curry states that most writers divide this disease into three classes or types: (1) *Pestis bubonica*, or bubonic plague; (2) *Pestis siderans*, or septicæmic plague; (3) *pulmonic plague*. Bubonic plague is by far the most frequent type. There is a type called *Pestis minor* (mild, or walking plague), with very low mortality, cases of which invariably precede epidemics. The invasion of plague is generally sudden, and is often accompanied by a chill, followed by high fever. The onset is quite similar to that of the malarial fevers. Rats are very susceptible to this disease, and often die in great numbers in the course of an epidemic; flies, ants and other insects act as carriers of the plague bacilli. The first and greatest prophylactic measure is cleanliness. Rats and mice should be destroyed. Food and drink should be protected from possible contamination. During epidemics all food should be cooked.—*American Medicine*, May 31st, 1902.

Business Notices.

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THE

Journal of Tropical Medicine

JUNE 16, 1902.

BRITISH MEDICAL ASSOCIATION.

Seventieth Annual Meeting, Manchester, July 29th, 30th, 31st, and August 1st, 1902.

Section Q.—TROPICAL DISEASES.

President: Sir WILLIAM R. KYNSEY, C.M.G.

Vice-Presidents: Prof. RUBERT BOYCE, M.B.

MAX F. SIMON, C.M.G., M.D.

DEAR SIR,—At the forthcoming meeting of the British Medical Association at Manchester it is hoped that the work of the Tropical Section will be as important and interesting as it has been at previous meetings, and we trust that you will assist the Executive to realise this hope.

The following subjects have been selected for discussion:—

Wednesday, July 30.—"Prophylaxis and Treatment of Beri-Beri." The discussion on this subject will be opened by Dr. P. Manson, C.M.G.

Thursday, July 31.—"Dysentery." The discussion on this subject will be opened by Dr. M. F. Simon.

Friday, August 1.—"Yellow Fever." The discussion will be opened by Mr. Cantlie.

The Section will meet on each day at 10 o'clock, when the above discussions will be immediately entered upon.

On Wednesday the business will be introduced by a short address from the President, Sir William R. Kynsey.

Drawings, photographs, card specimens, microscopic preparations, or lantern demonstrations illustrative of disease, are always instructive and never fail to excite interest. Those concerned in the work of the Tropical Section are requested to help in this direction.

The following gentlemen have already intimated their intention to take part in the discussions, or have promised papers: Dr. Patrick Manson, Mr. James Cantlie, Major Ross, Dr. M. F. Simon, Dr. Edward Henderson.

Papers have been promised by: Dr. Andrew Duncan, Mr. James Cantlie, Prof. Hewlett, Mr. P. W. Bassett-Smith, Dr. J. Galloway.

We shall be obliged if you will let us know, at your early convenience, if you will be able to take part in any of the above discussions, or if it is your intention to contribute a paper on any subject within the range of the Tropical Section.

It is obvious that presence in England is necessary for a member to take part in person in the discussions, but we would point out that this is by no means necessary as regards papers either containing observations on the subjects to be discussed, or on other subjects, which may be sent to us to be read at the meeting.

We are, dear Sir,

Your obedient Servants,

BRIAN MELLAND, *Hon. Sec.*,

Ashley Road, Bowdon.

C. W. DANIELS, *Hon. Sec.*,

Seamen's Hospital,

Albert Dock, London, E.

March 25th, 1902.

Extract from Regulations for the conduct of Annual Meetings of the British Medical Association.

(1) Papers at the Sectional Meetings must not occupy more than fifteen minutes in reading, and no subsequent speech must exceed ten minutes.

(2) Authors are requested to send short abstracts of their papers not later than Saturday, June 28th.

DR. P. T. MANSON'S FUNERAL IN HONG KONG.

On the evening of May 5th, the remains of Dr. Patrick Thurburn Manson, eldest son of Dr. Patrick Manson, C.M.G., Medical Adviser to the Colonial Office, were laid to rest in the Happy Valley Cemetery in the same grave in which was buried a younger son of Dr. Manson, who died here in 1887.

The immediate mourners were Sir Francis Lovell, C.M.G., and Professor Simpson, representing the Tropical School of Medicine, Drs. Hartigan and Rennie, partners of the firm to which Dr. Manson formerly belonged, and Drs. Atkinson and Laing, both of whom were personally acquainted with the deceased. The Rev. T. W. Pearce read the service, the first part being held in the mortuary chapel. The body was

borne to the grave from the chapel by eight of the members of the European Police Force.

Amongst those present were: Sir Thomas Jackson, His Honour A. G. Wise, Hon. T. H. Whitehead, Mr. F. A. Hazeland, Drs. Harston, Clark, and Gibson, Messrs. T. E. Cocker, H. N. Mody, B. Layton, W. H. Ray, J. H. Cox, D. B. Law, H. W. Robertson, G. A. Caldwell, F. Maitland (Messrs. Linstead and Davis), and many other old residents.

Wreaths and crosses were sent by the following: Major-General Sir Wm. Gascoigne, K.C.M.G., Mr. and Mrs. Wise, Mr. T. E. Cocker, Dr. and Mrs. Atkinson, Mr. and Mrs. F. Maitland, Dr. Noble, Mr. and Mrs. A. G. Gordon, Madame Rieco, Mr. and Mrs. G. Geiger, Mr. H. N. Mody, Mr. and Mrs. Layton, Dr. and Mrs. Hartigan, Dr. and Mrs. Laing, Dr. L. P. Marques, Mr. and Mrs. Bell-Irving, Dr. and Mrs. Stedman, and Mr. Morehead. There were also a number of wreaths which had no card attached.—From *Hong Kong Daily Press*.

AN APOLOGY TO THE "JOURNAL OF HYGIENE."

WE owe an apology to the *Journal of Hygiene* for inserting in our issues of May 1st and May 15th an article on "Recent Researches Concerning the Etiology, Propagation, and Prevention of Yellow Fever by the United States Army Commission, by Walter Reed, M.D." We were so impressed with the value of the article, that we resolved to insert the article *verbatim*. We duly acknowledged the source of the article, but we omitted to obtain the permission of the Editors or publishers to insert it. For this omission we humbly and readily apologise. We regret the incident extremely. The *Journal of Hygiene* stands so pre-eminent amongst medical journals in Great Britain that in any way to injure it would be nothing short of a national offence. We have no other journal in the medical literature of this country that takes the same standing, or that devotes itself solely to recording and dealing with research in its widest meaning. Having these opinions, it is scarcely to be thought of that we copied into the JOURNAL OF TROPICAL MEDICINE any article appearing in the *Journal of Hygiene* with the idea of inflicting injury. The *Journal of Hygiene* has been instituted but quite recently, and although its reputation is widespread, we thought it becoming in the interests of the *Journal of Hygiene* to let its excellent publications be known as widely as possible. It was in this spirit that we endeavoured in our humble way to benefit the *Journal of Hygiene*, but it appears we were inflicting injury by so doing, and for this we wish to communicate our regrets and apologies.

A CASE of plague was reported on board the P. & O. steamer *Victoria* from Bombay. The man was stated to have thrown himself overboard. No other case occurred. At Plymouth the passengers and crew were inspected, and the names of those landing were taken.

Review.

MILITARY OPHTHALMOLOGY. A Manual for the Use of Medical Officers of the Home, Indian and Colonial Services. By M. T. Yarr, F.R.C.S.I., Major R.A.M.C. Cassell and Co., Ltd. London, 1902. With diagrams. Pp. 236.

Major Yarr's name is so well known as an authority on Ophthalmology, especially in the departments of tropical affections of the eyes and of all appertaining to military requirements, that we welcome the manual he has produced as emanating from one of our best, if not indeed our best authority on the subjects he has made specially his own. The manual contains not only instructions in vision-testing and estimation of errors in refraction, but also places clearly before us such subjects as malarial eye affections, eye injuries, venereal damage to the eyes, malingering, and a mass of useful information pertinent to the wants of medical men in the public services and to practitioners resident in warm climates.

The text is apportioned in fifteen chapters, of which No. ix., Malarial Affection of the Eye and Quinine Amaurosis, No. x., Tropical Eye Diseases (non-malarial), and No. xi., Trachoma (Military Ophthalmia), will prove specially interesting to tropical practitioners. We have been favoured by Major Yarr with so many valuable articles on these subjects in the JOURNAL OF TROPICAL MEDICINE, that the readers of the Journal are no doubt acquainted with some of the more important facts brought forward in the manual; nevertheless, to have them collated, amplified and systematised as they now are, will be a keen satisfaction. A most valuable appendix deals with "The Vision Regulations of the English [British?] Army compared with those of other Armies."

The whole manual is written in a style which commends itself by its precision, its thoroughness and its clearness. The chapter on Operations is a model which other writers would do well to copy.

We congratulate Major Yarr on his book, and we expect to see the manual he has written in the hands of every medical man leaving these shores to take up work abroad whether in a civil or military capacity.

PREVENTIVE HYGIENE.—Those interested in the subject of venereal disease and its prevention will find the question dealt with in a pamphlet on "The International Conference held at Brussels," September, 1899, written by an English member of the Conference. A second edition has been recently issued, and may be obtained from Pewtress and Co., 28, Little Queen Street, Lincoln's Inn Fields, London. Price 3d.

FOR DYSENTERY.

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|---|-------------------------|-----|-----|-----|---------|
| R | Acidi sulphurici diluti | ... | ... | ... | 3ss. |
| | Tinct. opii deodorati | ... | ... | ... | |
| | Spt. camphoræ | ... | ... | ... | āā 3j. |
| | Tinct. capsici | ... | ... | ... | |
| | Spt. chloroformi | ... | ... | ... | āā 3ss. |
| | Spt. vini gallici | ... | ... | ... | 3jss. |

M.S.—5j. q. 2—3 h.—*The Medical Times*.

News and Notes.

SORCERY, MEDICINE, AND SURGERY IN ANCIENT MEXICO. By ZELIA NUTTALL.

In the Johns Hopkins Hospital Bulletin for April, 1902, Zelia Nuttall gives an interesting account of ancient Mexican medicine men and their **superstitions**. Many of the customs, &c., described are, of course, common to all mankind; nevertheless some interesting descriptions are given by the authoress showing the "curious mixture of ignorance and practical common sense" which attaches to most of the necromantic practices in vogue amongst primitive peoples. Some of the practical hygienic details are especially interesting. In regard to the **preservation of the teeth** is it recommended: (1) To avoid eating very hot food; (2) not to drink cold water immediately afterwards; (3) clean the teeth after eating with a wooden toothpick; (4) "use cold water and salt for cleaning the teeth, and rub them frequently with a cloth and some finely-ground charcoal."

"The sweat-house" was regarded by the ancient Mexicans as invaluable in many ailments.

Massage for rheumatic pains, sprains, and even a cough is highly extolled.

Nose bleeding in the year 1576 amounted to a pestilence; it is referred to as being cured by holding a bloodstone, but what the exact nature of the ailment with which nose bleeding was associated is not revealed.

Native herbs and their properties were well known in ancient Mexico, and near Monte Zinna's palace a garden was specially set aside for the cultivation of medicinal herbs.

The authoress has given us most interesting information, and we hope that this is but an introduction to the folk and medical lore of the interesting people who inhabited Mexico in early times.

up the water like a sponge. The emptied vascular system is refilled, blood pressure is raised, and the heart regains its vigour. The child will probably fight shy of the first teaspoonful, fearing probably it is beef-juice or medicine, but the greed with which the next and all water given up to a half pint is taken, the satisfaction, the calm, and the sleep which ensues, show how badly water is needed.

"Food and 'stimulants' are harmful; if they are retained, attempts at digestion and absorption take place. Water, which should be boiled, supplies all that is essential. The mucous membrane rests and recuperates, so that after twelve to twenty-four hours it is fit to resume light work. Moreover, in addition to relieving urgent symptoms, the water is directly curative; it dissolves the absorbed toxins, the kidneys again become active and eliminate them. The intestinal bacteria die from inanition; their pabulum is cut off. When the urgent symptoms have ceased—as they almost invariably will do in twelve to twenty-four hours—milk may be given in very dilute form; water must yet constitute the chief aliment. For the first day one part of milk to twenty of water is given. From day to day the amount of milk is increased until the normal proportion is reached. The exclusively water diet may be continued with safety up to forty-eight hours, but only very rarely is it required after twenty-four hours, when diarrhoea and vomiting have ceased. Tepid sponging if there is fever, hot, dry packs if there is collapse, and large *linseed* meal poultices to cover the abdomen if there is pain, are valuable auxiliaries.

"In addition to the above I would like to suggest the advantages to be derived from washing out the stomach through a tube passed through the nose, and from the use of large water enemata. These allow absorption and at the same time remove the toxins."

ULCERS OF THE LEG.—Dr. O. Schulze (*Münch Med. Woch.*, March 19th, 1901), says that of all remedies, new and old, camphor gives the best results in ulcers of the leg. His prescription is as follows:—

| | | | | | | |
|-----|---------------|-----|-----|-----|-----|-------|
| R | Camphor trit. | ... | ... | ... | dr. | ss. |
| | Zinc oxid. | ... | ... | ... | „ | viii. |
| | Adeps ad. | ... | ... | ... | oz. | vi. |
| Or, | Camphor trit. | ... | ... | ... | oz. | ss. |
| | Zinc oxid. | ... | ... | ... | „ | iii. |
| | Olei olive | ... | ... | ... | „ | iii. |

Current Literature.

TREATMENT OF SUMMER DIARRHOEA IN INFANTS BY EXCLUSIVELY WATER DIET.—In the *Clinical Journal* of July, 1901, Dr. J. D. Windle advocates a "water" diet for young children in the tropics suffering from summer diarrhoea. In his article Dr. Windle expresses himself as follows:—

"The amount of water lost in the stools and by vomiting and perspiration is out of proportion to that taken; in most cases none is absorbed, for vomiting is constant. The excess must come from the blood and tissues. Thirst is the cause of the restlessness and moaning symptoms often wrongly interpreted as due to pain. The great loss of weight, sunken fontanelles, flaccid skin, scanty micturition, dyspnoea, and cyanosis, are due to the dryness of the blood and tissues. The vital indication is to supply water, which at once relieves the most urgent symptoms—thirst and vomiting. The blood-vessels of the stomach suck

PRESCRIPTIONS.

TOOTHACHE.—Place in the painful cavity a plug of cotton wool soaked in

| | | | | | | |
|---|---------------|-----|-----|-----|-----|-------------------|
| R | Codeine | ... | ... | ... | ... | $\frac{1}{4}$ gr. |
| | Oil of cloves | ... | ... | ... | ... | 3ss. |
| | Chloroform | ... | ... | ... | ... | 3iss. |

VOMITING OF PREGNANCY:—

| | | | | | | |
|---|----------------|-----|-----|-----|-----|----------|
| R | Menthol | ... | ... | ... | ... | 2 parts. |
| | Ol. oliv. opt. | ... | ... | ... | ... | 10 " |

M.S.—Ten drops in sugar when nausea occurs.—*The Medical Times*.

PLAGUE.

PREVALENCE OF THE DISEASE.

INDIA.—During the two weeks ending May 10th and May 18th the number of deaths from plague in India numbered 11,612 and 7,008 respectively. During the two weeks in question the deaths from plague in the principal centres of the disease were as follows: Bombay City, 391 and 300; Bombay districts, 583 and 429; Calcutta, 280 and 209; Bengal 337 and 136; United Provinces, 401 and 266; the Punjab, 9,192 and 5,453.

EGYPT.—During the two weeks ending May 18th and 25th the numbers of fresh cases of plague reported in Egypt were 33 and 26; the deaths during the same period amounted to 28 and 16 respectively. The towns and districts in which plague occurred were Alexandria, Mit-Ghamr, Mit-Samanoud, Dechneh, Tala, Maghagha, Toukh, Samalouh, Menouf, Beni-Mazar, Damietta, Achmoun and Damanhour. In none of these places, however, has plague obtained a serious hold.

HONG KONG.—During the weeks ending May 27th and June 3rd the number of fresh cases of plague in Hong Kong amounted to 33 and 52, and the deaths from the disease to 33 and 50 respectively.

CAPE OF GOOD HOPE.—During the week ending May 10th two fresh cases of plague occurred at Port Elizabeth and three deaths from the disease.

A PHENOMENON OBSERVED ON THE TONGUE IN ACUTE MALARIAL INFECTION. Dr. Lucien Lofton, in the *New York Medical Journal* of April 12th, 1902, states:—The condition of the tongue that I wish to bring out in connection with acute malarial poisoning is nearly always present, more or less, and may be said to be pathognomonic of the disease. This condition presents, upon exhibition of the tongue, one or more (generally two) dark lines running from the base of the organ to the apex, and usually separated by a clearly-defined tract of clean mucous membrane about one-sixteenth to one-eighth of an inch wide. These lines are pyramidal in appearance, and begin among the large papillæ at the base of the tongue. They vary in width, and may be from one-eighth to one-quarter of an inch wide, gradually coming to a point in the middle of the tongue. In colour they resemble the stain of a 10 per cent. solution of potassium permanganate that has been exposed to air for some time. This condition will most likely be found from one day to two weeks after exposure or inoculation. It remains in some individuals longer than in others, notably in the negro. It is more beautifully defined from six to twelve hours after the initial sporulation, and remains until the system is thoroughly cinchonised. Purgation alone, so far, has failed to relieve the condition, for the gamut of drugs has been run in this connection time and again. Only one line will be found more often in the coloured man than in the white.

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Archives Russes de Pathologie, de Médec. Clinique et de Bacteriologie.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Brooklyn Medical Journal.
Caducée.
Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Esercito.
Hong Kong Telegraph.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
Journal of the American Medical Association.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Medical Record.
Medical Review.
Merck's Archives.
New York Medical Journal.
New York Post-Graduate.
Pacific Medical Journal.
Polyclinic.
Public Health.
Revista de Medicina Tropical.
Revista Medica de S. Paulo.
Sei-i-Kwai Medical Journal.
The Hospital.
The Northumberland and Durham Medical Journal.
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The Journal of Tropical Medicine.

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In order to meet the constant enquiry for addresses of respectable firms catering for the various requirements so difficult to obtain abroad, we give a list of names and addresses which we trust will be found useful to our numerous correspondents and subscribers.

Original Communications.

EPIDEMIC DYSENTERY IN GRENADA DURING THE LATTER MONTHS OF THE YEAR 1901. A CONSIDERATION OF ITS CAUSE, SYMPTOMS, AND TREATMENT.¹

By A. B. DUPREY, M.R.C.S., L.R.C.P.

Colonial Assistant Surgeon, St. Lucia; late District Medical Officer, Grenada.

IN as much as I have been asked to give a report of this present epidemic of dysentery now raging through all the districts of the Island of Grenada, I shall endeavour, to the best of my ability, to give in as precise a manner as possible my impressions, and the observations which force themselves upon me, and the course of action and treatment I adopted during my rounds of practice among the people. In a district of such a size and population, being at least twelve miles by six, and possessing not less than ten thousand people, it is well nigh impossible for any one man to do more than give his attention to each patient in succession, and to point out to the attendants the proper course and advice to be followed for the successful management of each individual case; for it may happen that the medical attendant will not have an opportunity of paying a second visit to any one case.

Dysentery is at all times a difficult disease to manage, and one which requires, even in hospital practice, all the care and attention of both medical man and nurse. What must it be, therefore, appearing as it has done for the last three or four months in epidemic form, and in such a district where some of the places are almost inaccessible? Any attempt at a scientific investigation by an officer during

the hurry of practice at such a time is, of course, out of the question, as can be well understood by any person who is thoroughly acquainted with the district; an honest and accurate study of each case as it presents itself is the only possible course, for as regards any elaborate clinical details, which can only be got by watching cases through their whole course of illness, one can hope to do so only in a very few instances.

The primitiveness and nonchalance of the peasantry in matters of sanitation is simply outrageous, and is hardly conceivable in these days, where the peoples of other nations are striving their utmost to arrive at, as near as possible, a state of perfection in matters appertaining to preventive medicine. The obstinacy of ignorance, even in some of the more educated classes, has, unfortunately, been the cause of many a fatal case, who otherwise might have recovered from this fearful malady; and not a few cases have succumbed to the more regrettable practices of some persons who pose as experts for this "belly complaint," which the easily gulled are told cannot be treated or even understood by the doctors.

CAUSATION.

The world of science has long ago recognised epidemic dysentery to be a water-borne disease, so one naturally turns to eliminate the first and most probable factor in the causation of the disease. This factor once made manifest, prevention easily follows. I personally have no doubt whatever that, were the water to be examined chemically and bacteriologically, the source of all this trouble would be found therein: a glass of water as obtained from the pipes of the town of Gouyare, if examined critically, even with the naked eye, cannot but suggest to the mind grave possibilities of infection. It is very turbid and rich in solids, has a sickly nauseous smell, even sometimes putrid, and decidedly heavy to the taste. It leaves a deposit on standing, and were it to be submitted to evaporation in the usual way would, I dare say, leave

¹ A paper prepared for the Government of Grenada, and published with the permission of the Colonial Surgeon.

an abundant residue, no doubt of the nature of a poisonous vegetable alkaloid. Knowing, therefore, that all epidemics of dysentery have been traced to an impure water supply, I have adopted the course of recommending to the inmates of each house visited the precaution of boiling their water and, if possible, filtering it also. This recommendation was, I am glad to say, promptly adopted in the parish of St. Marks, where the epidemic lasted but for a short time. It was short and sharp, forty-three cases receiving medical aid in the course of a few weeks. In the parish of St. John's, however, the cause of the epidemic was generally believed to be in the air, which belief obtained not only among the lower classes, who were practically the only sufferers, but even among some of the better educated, who, although dogmatic on the point, yet took the precaution necessary to safeguard themselves against infection by boiling and filtering their water, or by the exclusive use of pure rain-water for drinking purposes. This obstinacy could not therefore have but one result, and that was disaster, for when the parish of St. Mark's was absolutely free of dysentery, St. John's, and especially the town of Gouyare still suffered, and likewise those other parishes whose people did not adopt the only preventive possible in such an emergency. The water supply of the town of Gouyare is obtained from a reservoir, which is unworthy of the name according to the modern principles of storage of water for the public use.

The year 1901, I am told, has been an unusual one in the records of Grenada as regards its rainfalls, and the rivers have been almost continually flooded. Fruit has been so abundant that it dropped and lay rotting in the river bed. The reservoir is unprotected, either by filtrage or otherwise, and is merely a receptacle through which the river runs, the water being distributed in its original state. I am fairly justified, therefore, in believing that the cause of this epidemic, occurring as it does only among a certain class of the population who use exclusively the reservoir water, and who are not liberally fed, is not far to seek.

SYMPTOMS.

The attacks of dysentery are very much less, both in number and severity, than was the case three months ago. The symptoms are much milder, and unless it occurs in a person advanced in age, or otherwise handicapped by previous diseases, the prognosis is always favourable. Recovery is now the usual result, providing the case has been seen and treated early; and, even without systematic medical treatment, many recover.

The disease usually commenced with griping abdominal pains of a very severe nature, persistent retching and vomiting, which often were uncontrollable, and the passing of frequent small stools, consisting of glairy mucus and blood, with much straining and tenesmus. Often the motions consisted of almost pure, frothy blood, though not in any large amount, in fact, never any more than about a teaspoonful at a time. The patient was frequently doubled up with pain and straining; cold, clammy perspiration would sometimes pour from him through the incessant and ineffectual attempts at defæcation. His face would be pinched, with a settled, anxious expression on it,

which, on the hope of speedy help, would show a sudden and brief relief. This relief, however, which I have often observed, is only momentary, and soon there was a return of all the symptoms, with apparently greater force; his hands would be cold and damp, his pulse small and frequent, running up from 120 to 140 beats in the minute, the temperature either normal or subnormal, the mouth dry, the tongue thickly furred, and the patient would call constantly for water, which he swallows down with an avidity painful to see, his hand trembling as the glass is carried with much hesitation to his mouth, so that he has to be helped in the effort. He then falls back in bed in a state of utter exhaustion and collapse. Such a picture of distress was fairly common in the early days of the epidemic, and it is no exaggeration to say that the sufferings of the victims were even far greater than I have attempted to paint them. The symptoms were those of a violent gastro-intestinal irritant, which I have no doubt it was. After a very short time, indeed, seventy-two hours at the most, the motions would change to frequent, dirty, offensive, reddish-brown stools, and soon to the beef-washings of gangrenous dysentery: finally exhaustion and death. Symptoms of a very urgent and far graver nature, however, were observed in a few cases, which, to my mind, strengthened the view I have adopted regarding the causation of the epidemic. Those were of an extremely violent gastro-intestinal nature, and not unlike the symptoms that are produced by alkaloid ptomaines, usually known as ptomaine poisoning. The patient would be taken quite suddenly with retching and uncontrollable vomiting and diarrhoea, the surface of the body would be cold and perspiring, he would complain of great thirst and dryness of the throat; the pulse small, weak and running, could hardly be counted; the respiration rapid and shallow, and before the objective symptoms of dysentery would develop themselves death would take place from exhaustion and general collapse. Symptoms thus varied considerably from the very slightest attack to the most distressing condition detailed above, thereby affording ample grounds for the belief that a subtle poison is the agent and principal factor, and that the symptoms varied according to the dose.

It will be noticed that I have not mentioned **pyrexia** among the urgent symptoms observed during the epidemic, for the very good reason that fever was never a usual accompaniment in an attack of dysentery. The temperature was generally normal, or even subnormal. In only twelve cases, or a percentage of 4.6, did the pyrexia required to be treated and quinine administered. The fever usually was of an intermittent type and bearing all the appearances of a malarial origin, quite distinct from the dysenteric condition, so that it seems the lowered vitality of the patient, which must of necessity follow on an attack of dysentery, brought out the attack of malaria, hitherto quiescent in the subject. Some were "fever subjects," and naturally required to be treated for such. In one case temperature remained obstinately high, registering at one time 105° F., notwithstanding quinine and baths. In this case nothing could be retained either in the stomach or rectum; uncontrollable retching and vomiting defied both hypo-

dermic injections of morphia or hot applications to the epigastrium; the dysentery became rapidly gangrenous, shreds of mucous membrane were frequently cast out from the rectum, and death took place seventy-two hours after the first onset of symptoms. Pyrexia was more often observed in children, though never to any serious extent. Temperatures of 101° or 102° F. were treated by baths and spongings, or more or less disregarded, while attention was directed to the more urgent abdominal symptoms. I cannot, therefore, say that pyrexia has been a very marked symptom during this epidemic of dysentery.

SEQUELÆ.

Under this head will be considered briefly some of the most important and immediate sequelæ following upon an attack of acute dysentery. As far as I know, the after and immediate effects of this disease upon a person who has passed through a sharp attack have not been sufficiently studied. Such sequelæ as contractions of cicatrices resulting in obstruction are but rare events, and liver abscesses may occur a long time after the attack, when possibly they may be due to other agents besides the poisons of dysentery. There are more important results requiring far more consideration, as upon their careful and accurate recognition depend the safety of the patients. Like diphtheria, the sequelæ may prove far more dangerous than the disease, and especially in dysentery the patient may be carried off from cardiac failure through a paralysis of its muscular walls, although the dysenteric process may seem to be on the mend. In three cases **acute œsophagitis** was a marked symptom and gave considerable trouble, while associated with that were usually **acute or subacute stomatitis**, so that the swallowing of even small quantities of liquid was accompanied with great difficulty and pain. In one case this was a serious complication and nearly caused the death of the patient. **Bright's disease** was a frequent sequela; in six cases the bloated face, œdema of the legs and albuminuria were very marked; in one case the condition was not treated for three months, and was so apparently advanced as to seem hopeless, yet he rallied after a month or two of treatment in such a marked manner that the change was surprising. It was more common in children showing clearly a lesser resisting power to the dysenteric poison in the course of excretion through the kidneys, but even in them the inflammation was transient and passed off after careful treatment. One must see, however, the extreme danger of a chronic inflammation of the kidneys persisting in a person whose vitality has been considerably depressed by a long attack of dysentery. **Peripheral neuritis** is another condition observed in those who suffered severely from dysentery. They complained of cramps and weakness of the legs, of numbness and tingling. The course of the sciatic nerve was more or less tender in some and in not a few sensation in the limbs was impaired. This condition was always present in both limbs and not due, as one might suppose, from lying constantly in one position. There were other troublesome conditions observed, such as **urticaria** and **pruritus**, which latter kept the patient awake night after night and thus helped to prolong convalescence.

TREATMENT.

I now come to consider the question of treatment, which is by no means an easy matter, considering both the nature of the country and a people whose prejudice against qualified opinion and legitimate medicine is somewhat extraordinary to a logical mind. On the other hand, any "bush medicines" or other nostrums recommended by friends are taken up with great promptitude and persevered in with a doggedness born of the greatest fortitude. Thus Chamberlain's diarrhœa mixture rapidly disappeared from the druggists' shops, and the stock in the whole island was, I believe, speedily exhausted. Then recourse was had to the bark of the sea-grape and the cashew, which were boiled together, including other bush remedies, to form a tisane, the nature of which was highly astringent. This mixture was kept "in the pot" boiling all day and drank in teacupful doses *ad lib.* This crude remedy, as it appears, was the very worst that could have been chosen, for with the exception of opium and morphia, all astringents were distinctly contraindicated in the early stages of an acute attack, inasmuch as they caked up and bound the bowels, thereby increasing to a great extent the absorption of the dysenteric poison.

There were in all 260 cases of dysentery treated between August 7th and December 29th, 1901, of which number 224 are recorded as giving positive results one way or the other, the remaining 36 being doubtful as to their termination. The number of cases treated with **sulphur and Dover's powder** were 193, and the method adopted was upon the lines recommended by Dr. G. E. Richmond (*Lancet*, June 15th, 1901), with a mortality of 8.4 per cent. This is not the first time I have used sulphur in the treatment of dysentery, having experimented with it at the Colonial Hospital, Port of Spain, Trinidad, more than four years ago; but the over-crowding state of the diarrhœa ward at that time was such that it was impossible to derive any good results from the drug. Thus it will be observed that sulphur in the treatment of dysentery has afforded ample satisfaction, notwithstanding the disadvantages of practice in such a district. In most cases the patients, owing to long distances, were seen twice, and a large number but once only. The immediate result of taking 20 grs. of sublim. sulph. combined with 5 to 10 grs. of pulv. ipecac. co. every four hours, was in all cases a stoppage of the blood and mucus after taking half a dozen powders. The straining was much lessened and the motions were less frequent and more fluid. The griping pains, however, continued just as severe, and it was remarkable that in women the most severe pain was situated over the hypogastrium, so that they believed that their wombs were affected and my diagnosis completely wrong. These severe griping pains, I concluded, were due to irregular peristalsis of the inflamed bowels, which subsided at once on giving morphia. The resulting diarrhœa could always be checked by the administration of bismuth and opium. Most of the patients as a rule took the sulphur very well, and themselves noticed the immediate good effect of the treatment; a few complained of severe retching, but as this was one of the most marked symptoms of the epidemic, the sulphur could not be

put down as being the cause. In conjunction with the powders, and in all cases where retching was severe, I recommended frequent sips of very cold water, and sometimes applied a mustard poultice on the epigastrium. The treatment of dysentery by sulphur, whether in an acute or chronic stage, deserves, therefore, the highest recommendation; especially is this the case in district practice. The difficulties of making people keep to a prescribed diet are, of course, obvious. I have known one patient who had a severe attack and recovered, although he fancied nothing else but plain boiled salt beef and potatoes, which the wife said he ate heartily.

Ipecacuanha gave less good results on account of the distress into which the patients were thrown owing to constant retching and vomiting; 20 to 30 grs., always preceded by a draught of opium or a hypodermic injection of morphia, was the mode of administration. In some cases the depression produced by ipecacuanha were so great that the patients refused to take it, saying that it aggravated their condition, but in six instances where opportunities were afforded of watching those cases ipecacuanha gave excellent results. Usually in adults $\frac{1}{2}$ gr. of morphia was injected either in the arm or epigastrium, and the powder was given just about the time that the patient feels he is going off to sleep; given in this way the powder is retained, and any subsequent dose does not generally require a repetition of any morphia. In children the following formula was found to be very useful, viz., hyd. cum cret. gr. i., pulv. ipecac. co. gr. ii., bism. subnit. gr. iii., sulph. sublim. ad. gr. xii., every six hours, or as thought fit by the medical attendant.

The **saline treatment** of dysentery was tried in several instances where the cases were seen very early, and where opportunities were got of revisiting the patients; but in districts where time and distance must be taken into consideration, the sulphur mode of treating epidemic dysentery is, I am convinced, the best means we possess at present.

THREE LECTURES ON BILHARZIA,

Delivered at Kasr-el-Ainy Hospital, Cairo.

By FRANK MILTON, M.R.C.S.

Surgeon to the Hospital.

LECTURE II.—(Continued from p. 192).

URINARY CALCULI.

STONE, although a fairly common complication in this stage of bilharzia, occurs more frequently and is altogether of more importance in the earliest stages of the disease. When stone is present in the later stages it is, as a rule, composed of phosphates, and very soft, and its presence is hardly noticed where the symptoms are already so severe. It very often happens that patches of roughened mucous membrane encrusted with phosphatic deposit give rise to more severe symptoms than true calculi themselves, and it takes some little practice and care to diagnose this condition from true calculus. Calculi which occur in the earlier stages of the disease are of much greater importance, as they then give rise to their characteristic

symptoms, and are capable of making their presence felt. There is no doubt that the majority of these stones owe their origin directly to the bilharzia, for in practically all the cases of stone we get at the hospital, amounting on an average to about 150 a year, bilharzia eggs are either still present in the urine, or there is a history of bilharzial disease within the period of time necessary for the formation of the calculus. The majority of the stones, too, are formed in the bladder itself, and have not descended from the kidney, being due to the deposition of its salts by the urine during its stay in the bladder under the influence of the diseased mucous membrane, and are not due to imperfect elimination by the kidneys; besides it has sometimes occurred that small clots and collections of bilharzia eggs have been found forming the nucleus of the stone. The symptoms caused by these calculi are overshadowed and masked by the symptoms due directly to the bilharzia; but the presence of the stone greatly increases the patient's sufferings; indeed, the relief experienced by the patients after lithotripsy is so great that as a rule they look upon themselves as cured, and decline to waste time over the treatment of their bilharzia.

When the disease is well advanced in the bladder, it would only be natural that neighbouring parts, such as the prostate gland and the seminal vesicles, should become implicated and give rise to symptoms; great stress is laid by some writers on the sufferings of patients due to affection of these parts, and they attribute irritation of the bladder, vesical tenesmus, and dysuria to their implication; but I doubt if, whilst the disease is in a sufficiently early stage to permit, as it were, a sifting out of symptoms and their attribution to the precise structure on whose implication they depend, the prostrate or seminal vesicles will ever be found very particularly involved; and when the disease is of sufficiently long standing to have caused secondary changes in or a continuous extension to these parts, I doubt if the symptoms to which such extension would give rise would be of sufficient severity to make themselves felt as distinct from the true bladder symptoms. The abnormal ejaculations and spermatorrhœa also spoken of by these writers, I have never heard complained of, although they are symptoms which native patients would be quick to notice and to attach importance to. When death occurs as the result of bilharzial disease of the bladder, it is brought about either by exhaustion from pain and want of rest, together with debility consequent on the constant hæmorrhage, aided by poisoning from the absorption of septic matter from the bladder; or it occurs as a consequence of extension of disease backwards to the kidneys, setting up pyonephrosis, pyæmia, or uræmia. Sufferers from bilharzia are very often the victims of other parasitic diseases, such as ankylostomiasis, which also contribute towards a fatal ending; but the bilharzia itself is quite equal, without extraneous aid, to the task of destroying its victims, which it does in a large number of cases, and with unspeakable torture.

URINARY FISTULÆ.

The symptoms due to the formation of urinary fistulæ will differ accordingly as to whether the fistula

originates in the roof or the floor of the urethra. In the case of fistulæ arising from the roof the symptoms are comparatively mild and unimportant, and in our hospital practice patients are seldom or never seen in the stage of formation of fistulæ the discomfort not being great enough to cause them to leave their work to seek relief. In the majority of cases it is probable that the symptoms in the early stages of the malady are so comparatively slight that they pass unregarded in the presence of the more severe suffering entailed by the disease existing in other parts of the urinary system; for I doubt if the urethra is ever attacked with bilharzia without the bladder being first implicated.

It is quite easy to understand, then, that in the presence of the irritation due to the passage of unhealthy urine along the urethra, the pain likely to be caused by a slowly forming ulcer or sinus would pass unnoticed, and this is probably nearly always the case. When, however, the process of disintegration approaches the skin surface, the disease begins to make its presence more acutely felt, for the vitality and resistance of the skin is so great that it cannot be destroyed until the accumulated products of destruction due to the progress of the disease have set up sufficient inflammation to cause the formation of a subcutaneous abscess, and it is a result of this suppuration that the skin is finally perforated and the fistula completed. The first symptom, then, which arrests the patients' attention in cases of roof fistulæ, is the formation of a subcutaneous abscess, generally in the perineum, which, after existing for a variable time, opens and discharges a small amount of unhealthy pus, which, if it were examined microscopically would be found to contain numerous bilharzia eggs. The abscess after it has opened and discharged its contents shows no inclination to heal, and the patient then notices that after micturition the amount of discharge is increased, and finally he discovers that the discharge from the sinus consists of pus, mixed to a greater or less extent with urine.

The symptoms due to fistulæ originating in the floor of the urethra are very much more severe than this. In the first place, as soon as the invaded mucous membrane has broken down and formed an ulcer, or rather, perhaps, as soon as this ulcer has begun to extend into the underlying tissue, the urine and the debris from the bladder becomes entrapped, as has been explained earlier, and forms a peri-urethral abscess, which is continually discharging its contents into the urethra. The actual pain in the abscess itself is not very great, but the inflammation set up in the delicate mucous membrane of the urethra by the irritating discharge is very great indeed, and the pain is increased by the passage of the urine over the inflamed tissue. Besides the pain and scalding due to the urethritis, there is always difficulty in micturating, for in the early stages the urethra is narrowed by spasm due to the pain, and later on true stricture is developed by the formation of inflammatory tissue in the whole length of the urethra on the distal side of the fistula. This stricture, if the inflammation is kept up for a length of time, may narrow the urethra down to a point where the finest instruments are unable to be passed along it, and the

urine can escape only in drops, the greater part passing through the fistula, and in a certain proportion of cases the urethral canal becomes obliterated altogether, and is merely represented by a solid cord of fibrous tissue. These strictures when once they are fairly established are extremely resistant to treatment by dilatation, for their extent is very great, and the new tissue is so dense and tough that it will not yield to any justifiable amount of force, and even if it can be made to stretch over the catheter, it will invariably return to its former condition on the instrument being withdrawn.

As I have already said, it is very rare for a man to come for treatment of a roof fistula before it has reached the stage at least of subcutaneous abscess, but with floor fistulæ it is very different, the majority of patients seeking relief even before the sinus has approached the skin surface, owing to their difficulty in passing water, and to the severe pain and scalding due to the presence of urethritis.

The signs of the two forms of fistulæ are as distinctly characteristic as their symptoms: Thus, in roof fistulæ the openings of one or more sinuses are found usually in the perineum or posterior aspect of the scrotum, or it may be in the anterior aspect of the scrotum, or on the pubes or thighs, but they are almost invariably away from the direct line of the urethra wherever they may be situated. The openings are small, with protruding granulations at their mouths, and the skin around is somewhat scarred and drawn in towards the opening as to a central point, and on manipulation a certain amount of thickening and matting together of the underlying structures can be felt, which is not very well defined and becomes more indefinite the deeper it is traced. In the floor fistulæ the signs are much more prominent. The openings, although generally in the perineum, are frequently in the penis anterior to the scrotum, and they are always in the immediate line of the urethra. The openings themselves resemble those of the roof fistulæ, but the skin is, as a rule, much more extensively adherent and thickened, and leading from the skin surface directly down to the urethra and clearly connected with it, will be found a sharply-defined mass of stony hardness and varying extent. The meatus urinarius will generally be found much narrowed and scarred, and probably thin, unhealthy pus will be found oozing from it, and on introducing a catheter a well-marked stricture will almost certainly be found. None of these latter signs are seen in roof fistulæ even of long standing, whereas they are almost constantly present in floor fistulæ, and the stony tumour connected with the urethra, with narrowing and discharge from the meatus, will be found even before the fistula has reached the surface and become complete.

BILHARZIA IN THE INTESTINE.

Bilharzia of the intestine, which, for our purpose, may be confined to bilharzia of the rectum, follows much the same course as bilharzia of the bladder, with its symptoms, of course, modified by the structure and functions of the part invaded. The earliest symptom of rectal bilharzia would appear to be not hæmorrhage, as in the case of the bladder, which

is there caused by rupture of superficial vessels by the mechanical force of the contracting viscus, but over-secretion of mucus from excess of function of the irritated glands in the hypertrophying mucous membrane, together with irritation in the part due to the presence of the numerous eggs. This excessive secretion of mucus and irritation give rise to sensations resembling those caused by the presence of faeces in the rectum, pressing down on to the sphincter, and causes a feeling of the desire to evacuate the bowel in the patient, who, in consequence, is constantly trying to obtain relief by attempts at defaecation; these repeated efforts end, as a rule, in nothing but the passage of small quantities of faecal matter enveloped in mucus, and this only after a great deal of straining and forced effort. The cause of the uneasiness not having been removed the patient experiences but little relief from his small evacuation, and is induced after an interval to try again to obtain a more satisfactory result. The constant efforts at evacuation, accompanied as they are with much straining, tend, after a time, to increase the already abnormal state of hyperæmia and irritability of the rectal mucous membrane, and it tends at the same time to cause relaxation of the sphincters and to induce a state of prolapse.

As the disease advances and the bilharzial infarction extends the mucous membrane becomes more hypertrophied and its secretion increased, and the superficial vessels become engorged and liable to rupture. The desire to empty the intestine, and its accompanying tenesmus, becomes more frequent and urgent, and the patient redoubles his efforts, with the result that he is now constantly called to stool and passes varying amounts of blood and mucus, mixed with small quantities of faecal matter, and, of course, including numbers of bilharzia eggs. This constitutes the so-called bilharzial dysentery. When the disease has reached this stage the mucous membrane will be found to have undergone permanent alteration in the way of overgrowth, hyperæmia, and probably the formation of polypoid adenomata. The frequent and continuous effects at expulsion increased by the development of these growths, and lasting for long periods, will at last cause the heavy, thickened mucous membrane to be itself protruded from the anus and the stage of prolapse is reached, which in its turn may progress, until the greater part of the rectum is constantly outside the anus. The prolapsed bowel can, at any rate at first, be easily returned by its own retraction; but after a time, as more bowel protrudes, the patient will find that he has to employ his hands to force the prolapsed part back; and eventually, if he survives long enough, either the gut will become so thickened that all his efforts at reduction will be unavailing, or else the sphincters will become gradually paralysed from overstretching, and although the prolapse can be made to return it cannot be retained, and it will remain permanently outside the body and liable to injury and ulceration.

The state of a patient with bad rectal bilharzia in its later stages is almost as miserable as that of one in the last stages of bilharzia of the bladder, and death is brought about in much the same way by want of

rest and exhaustion, due to the constant efforts at defaecation and the never-ending discharge of mucus and blood.

Of course the whole progress of the disease is essentially chronic in its nature, and the patient may suffer for months and even years from the "dysenteric" symptoms before even prolapse occurs; and again, he may endure the sufferings incidental to this stage of the disease for an equally long period of time before death relieves him of his misery.

BILHARZIA IN THE VAGINA.

The symptoms due to bilharzial affection of the vagina are, on the whole, those of subacute vaginitis, but as the cases that I have seen have been so few in number, and have been so modified by treatment before coming to me, I feel I must wait for further experience and the examination of more unmodified cases before I can venture to tell you much about the disease in this locality. The only typical thing that I have so far recognised as likely to help you in your diagnosis is the very great thickening of the mucous membrane, especially on the posterior wall, and the peculiar and well-marked crossing fissures on its surface.

DIAGNOSIS OF BILHARZIA.

The consideration of the diagnosis of bilharzia need not detain us very long, for this depends essentially on the discovery of bilharzia eggs in the excretions of the diseased part or in the tissues of the part itself. In bilharzia in the *bladder*, the hæmaturia, with its peculiar nature and time of occurrence, namely, in the last few drops of urine passed, cannot be mistaken for that due to any other disease; the only difficulty likely to arise in these cases is to decide whether the patient has a stone in his bladder as well as a deposit of eggs, and this is at once determined on the passage of a sound. The determination in the later stages of the disease as to whether malignant growth has been grafted on to the bilharzial growth in the bladder is not, I think, of very great importance, and can only be determined with any degree of certainty by a digital examination of the bladder, except, perhaps, in those cases where the malignant growth is so extensive that it has overstepped the limits of the bladder and has involved the surrounding structures, when the nature of the case will at once be evident. As regards the *rectum*, the diagnosis is also, as a rule, quite simple, for although the earlier symptoms to some extent resemble those of chronic dysentery, still there are marked differences in bilharzia. The onset is invariably insidious, beginning simply with a sense of discomfort in the rectum, gradually increasing, and with the occurrence in process of time of frequency of defaecation, with straining and passing of mucus and some hæmorrhage, but this only develops very gradually, and never has an acute onset, as is usual with true dysentery. Again, there is never any general gastro-intestinal disturbance, such as expresses itself in true dysentery by the condition of the tongue, the severe abdominal pain, and fever; nor, as a rule, are the stools particularly offensive, and they do not contain visible sloughs; and where the disease is well established all doubt

in the diagnosis will be set at rest by the introduction of the finger into the rectum, and the discovery of the typical polypoid tumours. The *urethral fistula*, as a rule, give rise to no difficulty in diagnosis, for in this country, at any rate, it is extremely rare to find any other form of urinary fistula except those due to bilharzia, and a history can always be obtained from these patients of preceding bilharzia of the bladder. There is one form of bilharzia disease which may be wrongly diagnosed, and that is commencing fistula arising from the floor of the urethra, and before it has opened externally, that is to say, whilst it is still in reality in the stage of peri-urethral abscess. This particular form of the disease I have over and over again seen diagnosed, and indeed in early days have myself diagnosed, as stone in the urethra, and until the true nature of the case has been demonstrated by operation, it is very hard to believe that the diagnosis is wrong; for the extreme hardness of the walls of the abscess, which can be picked up between the finger, and can be felt to be intimately connected with the urethra, gives exactly the sensation of a stone obstructing and partly extruded from the urethra. When, moreover, the patient himself comes for difficulty in micturition, with pain and sense of stoppage at the site of this tumour, the diagnosis seems complete and the case comes to be regarded as one of long-standing, incomplete obstruction of urethra, due to the impaction of a calculus such as is so common among our out-patients. There are, however, two, or probably three, almost infallible signs which will put you on the right track, the first is the continuous escape of the unhealthy pus from the meatus, of which, by the bye, the patient seldom takes any notice; the scarred, glazed and narrowed look of the meatus itself; and the hard indurated feel of the urethra from the situation of the tumour right up to the meatus. Very often, too, it is impossible by squeezing the tumour firmly between your finger and thumb to cause an increased flow of pus along the urethra, and if then, to confirm your opinion, you pass a sound carefully down the urethra, your diagnosis will be completed.

I do not quite know how you would at first sight diagnose bilharzia of the *vagina*, but if you come across any obstinate cases of long-standing, subacute vaginitis with much thickening, and possibly fissuring, of the mucous membrane, I should advise you strongly to snip off a small piece of the membrane with a pair of scissors and examine it under the microscope for bilharzia eggs.

(To be continued).

CORONATION HONOURS.

K.C.M.G.

Surgeon-General H. Pinching, Head of the Sanitary Department at Cairo.

C.B.

Ronald Ross, Esq., F.R.S., F.R.C.S.

C.M.G.

Ho Kai, Esq., Unofficial Member of the Legislative Council of the Colony of Hong Kong. [Ho Kai is an M.B., C.M.(Aberd.), M.R.C.S.(England).]

We congratulate these gentlemen on their well-deserved honours.

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THE

Journal of Tropical Medicine

JULY 1, 1902.

THE LONDON SCHOOL OF TROPICAL MEDICINE AND SIR FRANCIS LOVELL IN THE FAR EAST.

ON the invitation of Dr. F. W. Clark, Medical Officer of Health for the Colony, a large number of medical men and members of the Press were invited to meet Sir Francis Lovell at dinner in the Hong Kong Hotel, on May 1st. The object of Dr. Clark's generous hospitality was to allow Sir Francis, who is travelling round the world in the interests of the London School of Tropical Medicine, to explain what are the objects of his mission.

In the course of his speech Sir Francis said: "The work that has been achieved in the School, as regards both research and ordinary clinical and bacteriological investigation, has been amply recognised by the profession, and the number of students has steadily increased. The present school buildings are far too small, whether from

a tutorial or from a research point of view, there is therefore imperative necessity for their extension. The laboratory and library should be doubled in size. Research laboratories, a lecture theatre, a mosquitorium and a museum are required. The residential accommodation should also be much increased; at present there is only room for six resident students; there should be accommodation for twenty. More travelling scholarships, similar to that generously endowed by Mr. J. C. Craggs, and known as the 'Craggs Research Scholarship,' are wanted.

"With these objects in view the Committee of Management, with the full concurrence of the Rt. Hon. the Secretary of State for the Colonies, resolved to accept my offer to proceed to India and the tropical colonies, especially where diseases peculiar to hot climates prevail, and to bring to the notice of the various Governments, and of influential and wealthy residents and employers of labour, the advantages they will derive from the School, and to endeavour to obtain their support for its extension. I accordingly started on this mission last October, and on my arrival at Bombay had to ascertain how I should proceed in the matter. At first I met with a good deal of discouragement. It was pointed out to me that things generally were in a bad way in that Presidency: plague and famine were prevalent; the cotton industry, almost the mainstay of the commercial world, was declining; many mills had already closed, and many mill-owners were bankrupt; calls for pecuniary help had recently been very frequent for local objects; and a list was then being circulated for subscription to the 'Victoria Memorial.' These were advanced as reasons why it was unlikely I should succeed with my mission in Bombay. However, after consultation with some of the leading members of the medical profession—European, Parsee, Mussulman and Hindoo—we decided to make an effort to obtain help for the School in that city. I commenced by giving an address on the subject of my mission to the members of the profession at the Bombay University, and after some debate, resolutions expressing approval of the scheme and

recommending it to the generous support of the Government and people of India were adopted. A small committee of medical gentlemen was appointed to collect subscriptions, and after a few weeks' stay in Bombay, I was able to remit to the bankers of the School such a sum as was very aptly described by the Committee of Management as constituting the result of my visit there phenomenal. Encouraged by this, my next visit was to Calcutta, where I spent the month of January; but there I did not meet with such support from the members of the Indian Medical Service as I did in Bombay, and on the whole my sojourn in that city was not as satisfactory as one would have expected. I received much sympathy with and encouragement in the object of my mission from some of the leading native medical gentlemen in Calcutta, and with their assistance succeeding in obtaining a few fairly handsome donations, and was able to bring to the notice of the public the advantages all residents in tropical countries are likely to derive from the work that is being carried on in the School. From Calcutta I proceeded to Ceylon, and there, again, I met with the same objections and difficulties that were said to exist in Bombay in successfully prosecuting my mission. I succeeded in obtaining from His Excellency the Governor the promise of an annual grant-in-aid for five years to the school on the condition that six students from Ceylon may be allowed every year to avail themselves of a course of study in the School without paying the usual fees. A Committee was appointed with Dr. Allan Perry, P.C.M.O., as Chairman and local representative of the School, and I am informed that in due course he has good reason to believe that substantial donations to the School will be forthcoming. I then proceeded to the Straits Settlements and Federated Malay States, and am glad to say that my visit there has proved very successful. His Excellency Sir Frank Swettenham, as Governor of the Straits Settlements and High Commissioner of the Federated Malay States, has promised the School an annual grant-in-aid of £200 for five years on conditions similar to those proposed by the Ceylon Government. His Excellency further

STUDENTS AND SOME OF THE STAFF OF THE LONDON SCHOOL OF TROPICAL MEDICINE.

9th Session, May-July, 1902.



G. F. Leicester, Lewis Savin, C. W. Brecks, W. Russell, A. A. Woods, E. J. Hynes, R. D. Clark, W. J. E. Davies, J. K. Reid,
A. Morley, H. F. Conyngham, C. F. Lasalle, L. E. Hertslet, A. E. Druitt, J. O. Summerhayes, G. D. Warren, P. Michelli (Secretary), C. W. Daniels (Med. Superintendent), A. Castellani,
Robert (2nd Lab. Assistant), Dr. W. G. Ross, Dr. Sambon, Miss H. M. Rowntree, Dr. Manson, Miss E. N. Tribe, Captain G. Hodgkinson, R. N. (Member of Committee),
Dr. M. F. Simon (Lecturer), Rev. W. E. Fay, G. C. Low (Cragg's Research Scholar).

decided to place the Directorship of the Kuala Lumpur Medical Research Institute in the hands of the Committee of Management of the London School of Tropical Medicine, who will in future select one of its students who has distinguished himself in research work and bestow upon him this post, which practically amounts to a scholarship tenable for three years, with an annual salary of £750, with free furnished quarters. I look upon this as the most important asset that the School has so far obtained. I travelled through the Federated Malay States, and succeeded in getting the support of the residents, from whom I received much valuable help; a committee, consisting of Mr. W. Cowan, Protector of Chinese, as Chairman, Drs. Wright and Edgar, and several leading members of the Chinese community, was appointed to collect subscriptions in the state of Perak, and up to the present very satisfactory results have been obtained. In Penang a similar course was followed, with His Honour Mr. Justice Leach, as Chairman, Mr. Hunter, Treasurer, and Dr. Freer, Colonial Surgeon, Secretary, and the news which I have already received from the latter is most encouraging. In Singapore, two committees have been appointed; one consists of a sub-committee of the Singapore Branch of the British Medical Association, with Dr. Galloway as Chairman; the other comprises four or five leading members of the Chinese community, and has for its Chairman the Hon. Mr. W. Evans, Protector of Chinese. Both these committees are doing all they can to further the objects of my mission and have already met with much success. I have now come to Hong Kong, gentlemen, and I hope that with your co-operation my efforts here to obtain support for the London School of Tropical Medicine will meet with at least as much success as they did in Singapore and its dependencies. I have hitherto found that it is most expedient to try and obtain the advice of my colleagues and others resident in any country that I may visit in regard to the manner in which I should proceed with my mission, rather than initiate my own manner of prosecuting it, and I propose, with your approval, to follow the same course in Hong Kong, and I shall therefore

conclude by asking you, gentlemen, to favour me with your views and advice."

Professor W. J. Simpson, Dr. Atkinson (Principal Civil Medical Officer), Hon. Dr. Ho Kai, Dr. Hartigan, and Dr. Rennie also addressed the meeting, and bore testimony to the good work done by the London School of Tropical Medicine. A small committee to act on behalf of the School, consisting of Drs. Ho Kai, Rennie and Atkinson, was appointed on the motion of Dr. Clark, seconded by Deputy Inspector-General Wright, R.N.

The School authorities in London owe a debt of gratitude to Dr. F. W. Clark for his timely assistance in affording Sir Francis Lovell so pleasant an occasion to bring forward the claims of their institution.

A PAPER ON YAWS.

By J. NUMA RAT, M.R.C.S.

Medical Officer, Leeward Islands, West Indies.

THE object of this paper is to summarise the additions made to our knowledge of yaws since 1891, and to deal with certain points regarding its nature which are still under discussion.

GEOGRAPHY.

The doubts entertained regarding the identity of the Paraji of Ceylon and the Coko of Fiji with yaws have been removed by the testimony of Sir William Kynsey and Dr. Daniels, and it is evident that the conclusion of Dr. Nicholls, that yaws did not exist among Asiatics to any great extent, was arrived at in ignorance of the fact that twelve years previous to the publication of his report in which this conclusion is expressed, Dr. Charlouis had described it as being widely prevalent among the Javans and Malays of the Indian Archipelago. Dr. Arthur Powell has also given us an account of an epidemic of the disease observed by him in Assam.

NOSOLOGY.

The position which yaws should occupy among classified diseases is a subject which has not received from recent writers the attention it certainly deserves. There was considerable diversity of opinion among observers at the end of the eighteenth and the beginning of the nineteenth century as to the true nature of the affection, some regarding it as one of the Cachexiæ, and others, as one of the Exanthemata. Hirsch (1885) describes it as distinguished from syphilis by having none of the properties of a constitutional disease, and having markedly the character of a local malady. Nicholls (1893) is of opinion that it may be either a local or a general disease.

Yaws is certainly not a cachectic disease. Like

syphilis it resembles the exanthemata, and, like that disease, it is capable of producing a cachetic condition of the system.

Those who consider it is a local malady only must ignore the constitutional symptoms that attend its development, and those who maintain that it may be either a local or a general disease base their opinion on the alleged absence in many cases of any constitutional disturbance. But the truth in this connection is that the experience on which statements with regard to its early symptoms are founded is very rarely personal. It is only after the general eruption has reached the fungating stage that patients generally come under the observation of medical men, and thus the conclusions of the profession relative to its preliminary constitutional effects are derived in the large majority of cases from the unreliable accounts of ignorant patients.

Inoculation experiments have supplied us with trustworthy information on this point. Those of Charlois show that in twenty-eight out of thirty-two persons inoculated there were "initial phenomena," comprising evening pyrexia, pains in the bones and gastric disturbance. But there is considerable difference in the statements of observers regarding the frequency of constitutional symptoms. Maxwell states that "yaws is generally preceded by symptoms of constitutional derangement." Bowerbank very briefly observes that after the incubative stage "there is an attack of feverishness with pains about the joints and shafts of the long bones." Charlois says "it usually commences with fever" and pains in the joints; Imray, that "as a general rule, there is very little, if any, constitutional disturbance either during the period of incubation, or on the accession of the eruption." Dr. Nicholls refers to the "difficulty in determining the exact train of symptom antecedent to and immediately following the evolution of the eruption, on account of the rarity of the cases which come under the observation of medical men in the earliest stage of the disease." "Frequently, however," he adds, "there are osteocopic pains, languor and debility."

"The invasion of the disease" he continues, "is doubtless in most instances marked by an elevation of temperature" which, however, may be so slight as to escape the attention of the patient. "In other instances, however, there is considerable constitutional disturbance which lasts from a few days to a week," and consists of continued pyrexia preceded by a rigor and the ordinary febrile phenomena.

It is evident from the above that further evidence is required to establish the alleged fact that yaws may develop without showing any constitutional symptoms and to prove that it may exist as a local malady only.

When yaws is attended by constitutional derangement, its course is like that of syphilis, and it should be classed with the latter.

HEREDITY.

Hirsch (1885) writes: "The opinions are divergent as to hereditary transmission of the disease (congenitally at all events, it seems never to have occurred, as there is not a single case of that kind given in the whole literature)."

No evidence has been adduced since 1885 to show that the disease has ever been seen at birth.

Whether it can be acquired in utero and manifest itself after birth is a question to which a definite answer cannot be given. It may be that yaws acquired in utero may be retarded, like syphilis, through several years, but, unless this is proved, we must conclude that the disease so contracted will reveal its presence not later than the end of the longest known period between inoculation and the appearance of the general eruption. Maxwell's experiments (1839) led him to believe that the interval between inoculation and the appearance of the general eruption varied from six weeks to three months, and he says that though there is considerable variation in this respect due to idiosyncrasy and the state of the patient's health, all observers agree that this interval did not exceed three months. Charlois (1881) gives the average period as three months, and, in some cases, four months.

Yaws, therefore (except as retarded yaws, if this ever occurs), cannot be considered to have been acquired in utero, if it should appear later than four months after birth.

But even in such a case there is the possibility of contagion during parturition to be taken into consideration. Maxwell refers to an attack of yaws in a child three months old (the earliest age at which he had known the disease to occur), which was supposed to have been contracted in utero, but with reference to which he suspected that "the mother had some remnant of yaws about the labia and that it had been communicated to the infant through the medium of a scratch in transitu." He considered that yaws was considerably modified during pregnancy and was never communicated to the foetus in utero.

It is possible that, as in syphilis, a child born of a mother who was suffering from recently contracted yaws during pregnancy inherits a certain morbid condition as the result of infection through the maternal blood, and that, instead of exhibiting all the phenomena of the disease, will, in course of time, be affected with some of the later manifestations only. This may, of course, be considered inheritance of the disease and may explain pathological conditions observed in persons who had apparently never suffered from yaws, which were attributed by some, in the early part of the last century, to leprosy, and by some, in modern times, to tuberculosis.

ETIOLOGY.

Nothing has been added since 1891 to our knowledge of the mode in which the disease is generally contracted. Its prevalence as an epidemic has suggested that yaws spreads by contagion, and possibly by infection. Inoculation experiments have confirmed the belief in its contagiousness, but we are still without any proof of its infectiousness.

Gavin Milroy (1873) questioned the contagious nature of yaws, or rather the truth of the belief that its prevalence was solely due to contagion, pointing out its frequency among the squalid and half-starved and its rarity among the cleanly and well-conditioned. Again, in 1879, he repeated his doubts on this point, and described the idea that contagion is the chief or

sole cause of the extension and propagation of the malady as "an unverified hypothesis."

This exemption of the cleanly and well-conditioned, even when they reside in the neighbourhood of those affected with yaws, or when, as in the case of doctors and nurses, they spend a considerable portion of their time under the same roof with yaws patients, leads to the inference that the disease is not infectious. It is also an argument in favour of its contagiousness, the immunity of the cleanly and well-conditioned being due to their avoidance of contagion, and the susceptibility of the squalid and half-starved to their uncleanness and their liability to contact with those affected with the disease.

Nicholls (1893) considers that "the examples showing the operation of contagion by contact are so numerous that this method of propagation must be taken as established beyond doubt." Numerous successful inoculation experiments have certainly been reported by observers at different periods, and these undoubtedly suffice to establish the fact that yaws can be contracted by contact of one of its cutaneous lesions with an abrasion of the skin if such contact be as intimate and prolonged as that which obtains in the case of inoculation experiments. But they do not prove more than this. They do not prove that the affection can be contracted by contagion during the ordinary intercourse of everyday life. To all accounts of cases in which it is declared to have been communicated by one person to another it can always be objected that the disease was caused in both by microbes in the air, or the soil, or the water.

The facility with which the disease can be acquired, whether it pass from one person to another or be caused by the germs in the soil, or the air, or the water, depends on the condition of the skin, and the affection will be most frequently found in places in which skin diseases are most prevalent. It is most probable that whenever yaws becomes epidemic some skin affection, such as itch, has been previously current in the locality.

Yaws is, generally speaking, far more common in the country than in the town, and while it may exist very extensively in some country district, there may not be a single case of it in a town only a few miles off. This is due in great measure to the fact, that country people (from the nature of their work in the fields) are more likely to suffer from injuries to the lower limbs than town people. It must be remembered also that the germs of the disease would find in the country conditions favourable to their development which are absent from towns, such as the shade and damp of deep valleys.

The above considerations lead me to believe that the affection in most cases originates with the entry of its germs from the soil into the skin. The amount of contact between individuals which is necessary for its transmission does not obtain in everyday life to the extent that would explain either its ordinary frequency or its periodical dissemination as an epidemic. But the development of its microbes in the soil and their specially rapid multiplication at a certain season would fully account for both.

Some observers have reported having found in the tissues micro-organisms which they consider to be the

cause of yaws. Pierrez (1890) was the first to report the existence of micrococci in the yaws tubercle. Nicholls (1893) also claims to have observed a micrococcus in the same. Breda (1895) gives an account of certain bacilli noticed by him in yaws tissues. Powell (1896) noticed in two cultivations from yaws a certain yeast whose cells lay in the granuloma. But in none of these cases has the microbe been proved to be the cause of the disease.

In a communication on the histo-pathology of yaws by Dr. J. M. H. MacLeod, Assistant to the Dermatological Department, Charing Cross Medical School, to the section of Dermatology of the British Medical Association, the following observations appear: "A search for a specific microbe in these sections proved futile. An examination for a new micro organism in sections of tissues is unsatisfactory at any time, and in the skin, this is even more markedly the case." . . . "We had no difficulty in finding micro-organisms in the horny layer in a number of sections which we stained in diverse ways for the purpose, but we only found types which could not be distinguished in their appearance from those which occur in the normal epidermis, such as cocci, diplococci, sarcinæ, and a few short bacilli. We failed to find the yeast fungus of Powell, and without cultivation experiments were unfortunately unable to verify the frambœsiform bacillus of Breda or the micrococcus of Nicholls and Watts."

While the micro-organism of yaws remains undiscovered, the possibility of a protozoic origin of the disease should not be forgotten. Neisser confirmed the opinion of Virchow, Klebs and Bollinger, that the tubercles of molluscum contagiosum were due to the invasion of the dermal cells by gregarinidæ, and it is interesting to note, in view of the liability of fowls to an eruption which is like that of yaws in appearance and which some consider to have a similar origin, that Bollinger, who described the "contagious epithelioma" of fowls in his *These de Paris*, 1860, maintains that this affection is identical with the molluscum contagiosum of man.

SYMPTOMATOLOGY.

Is there an **initial lesion** in yaws? Dr. Nicholls, on page 342 of his report of 1893, writes: "This matter has been alluded to in former places in this report, and I believe it has been proved that in the majority of instances yaws does not acknowledge a primary sore."

Tilbury Fox, in his work on skin diseases, writes as follows on the subject of inoculation: "Dr. Bowerbank tells me that if a poisoned wound be slight then little or no irritation may result and the part heals. But in other instances of inoculation the wound inflames and is covered with a brownish scab, beneath which is a small sore depressed in its centre and with raised everted edges and giving out ichor. This ulcer may heal up before the general eruption appears, but if large, this does not happen. In any case the primary ulcer becomes unhealthy when the general eruption appears and then fungated."

A similar account of what Dr. Bowerbank calls above "the primary ulcer" is given under the description of "Frambœsia," by Dr. James Christie, on page

395 of McCall Anderson's "Treatise on Diseases of the Skin" (1887), who refers to the ulcer at the site of inoculation as "the primary sore."

Charlouis mentions, on pages 306 and 307 of the New Sydenham Society's translation (1897) of his report on yaws, three cases of inoculation with yaws, in each of which an ulcer developed at the site of inoculation; three others on page 310, with similar results; and, referring on page 311 to the inoculations practised by him on 32 convicts, states that only four of them failed and, with regard to the rest, that, "after the lapse of fourteen days after the inoculation small papules were always seen at the seat of injection." These papules became pustular at twenty days from the inoculation and finally developed into deep ulcers with mottled floors and undermined and thickened edges. He also reports, on page 313, similar ulcers formed in connection with seven other inoculations. He further writes: "In the majority of patients the earliest appearances of frambœsia showed themselves after three months, but in some instances after four months." The lesion at the site of the inoculation occurred "after the lapse of fourteen days after the inoculation," so that the general eruption did not appear until more than two months after the lesion. Should this, therefore, not be termed a primary lesion?

That this primary lesion is not generally observed in persons who have contracted yaws in the ordinary way may be easily explained by the facts that yaws patients are very seldom seen by medical men before the appearance of the general eruption, and that the virus usually enters through an ulcer on the foot or the leg. Even when such a primary lesion exists the patient would not connect it with the general eruption that follows. Ulcers are so common among negroes of the labouring class in places in which yaws prevails that but little notice is taken of so small a lesion. It is very seldom, indeed, that I have seen a chancre in a black man; and Dr. Nicholls records a similar experience on page 217 of his report of 1893. "It is extremely rare," he writes, "for the labouring classes in the West Indies to apply for medical aid in the treatment of primary syphilis. The lesion, indeed, is either overlooked or made light of, for the people are ignorant of its consequences and are unwilling to bring to the notice of a medical officer what they would consider so trifling a thing as a little hard lump or a little sore. Thus, in my own practice in Dominica, I have rarely met with primary syphilis, but the secondary and tertiary stages are fairly common." Such a fact might lead anyone to consider whether a primary lesion in yaws might not be equally overlooked or made light of.

Dr. Nicholls's statement that, "should there be any growth at the site of the implantation of the virus, it differs in no essential particular from the characteristic eruption," is refuted by the results of Charlouis's experiments in which an ulcer developed at the inoculated point in every successful case. His argument that, as the growth at the site of inoculation is like the general eruption, therefore the former cannot be considered a primary sore, even if it were true, is one which might be similarly employed with regard to syphilis. For, if an erythematous eruption followed

an erythematous patch, or a primary lesion of any kind was succeeded by a general eruption of a like nature in a patient inoculated with syphilis, would it be wrong to call the local lesion a primary lesion?

When the primary sore of yaws fungates it does so only when it has lasted until the development of the general eruption, when the congested state of the skin at that particular point would necessarily be most favourable for the manifestations of the general infection.

It is evident that similar objections may be raised in other respects to the existence of a primary stage in syphilis. Even in venereal syphilis the primary lesion may escape detection, while in syphilis insonitum it is seldom that it is recognised as such, and, when so recognised, it is most probably entirely uncharacteristic.

Dr. Nicholls refers to his inoculation experiments, reported on pages 244, 245, and 246 of his report of 1893, in proof of his statement that there is no primary lesion in yaws. These experiments were performed under conditions which render the value of their results extremely doubtful. The conclusions he has drawn from them are certainly very different from those to which the numerous experiments of Charlouis and other observers unmistakably point. In only two of the cases were the inoculations performed by him in healthy individuals, that is, in those who had not suffered from yaws or who were not actually suffering from the disease. In one of these two cases a "pimple" appeared at the site of inoculation and developed into a tubercle, which disappeared by absorption, and a similar result followed in the second case. With respect to the other cases, there were lesions at the points inoculated in three instances. So that even as regards these inoculations, in five out of the eight persons inoculated there was evidence of a morbid process at the site of experiment. That a yaws tubercle developed at the point of entrance of the virus in those cases in which the persons were actually suffering or had previously suffered from yaws is easily explained by the existence of a general infection, which naturally produced its effects at the irritated point and modified the primary lesion.

But whether the morbid changes observed at the points at which the virus was inserted by Dr. Nicholls in his experiments were due to the action of the yaws virus introduced by him, or whether these persons were accidentally infected at those points by the microbes in the yaws hospital, or whether they only manifested symptoms of the disease from which they were already suffering, these are all questions which appear to me to be unanswered. Whatever may be the value of his inoculation experiments, those of Charlouis are a complete refutation of the statement that there is no primary lesion in yaws.

Exception has been taken by Dr. Nicholls to the statement that there is an **erythematous or scaly eruption** in yaws. The expression "erythematous or scaly" was used by Mr. Hutchinson in his preface to my essay, but I myself stated that minute red spots preceded the appearance of the papular eruption. "I must at once take exception to the accuracy of these terms as applied to the eruptions of yaws," writes Dr. Nicholls, on page 342. He then proceeds

to explain as follows: "The eruption which I have described as squamous is, of course, a scaly one, inasmuch as there is exfoliation of small flakes of necrosed epidermis. But partial or general desquamation is characteristic of many skin diseases, and it is simply an exaggeration of a physiological process that is always in operation in healthy individuals." He admits that he has applied the term "scaly" to a certain eruption which he has described as observed in yaws patients, but he takes exception to the accuracy of the term as applied to the eruption of yaws. He further writes on page 285 of his report, "The squamous patches not only occur at an early stage of the disease, but they may persist throughout the attack, or appear as a distinct eruption at any period of its progress." The contradiction involved in these quotations is evident. Maxwell describes a scaly eruption in yaws "not unlike pityriasis versicolor" as one of the precursive eruptions in yaws, and there is undoubtedly a papulo-squamous eruption which sometimes persists long after the disappearance of the general eruption, especially about the elbows and knees, where they simulate psoriasis.

With regard to the "erythematous" eruption, I would point out that Maxwell, in reference to what he terms the second variety of precursive eruptions in yaws, alludes to "the appearance on various parts of the body of numerous smooth, ovoid or circular blotches of a dark brown or dull reddish colour," and Bowerbank, in Gavin Milroy's report, page 55, gives the following account of the eruption which precedes the appearance of the yaws tubercles and follows the initial constitutional disturbance: "These symptoms" (those of the constitutional derangement) "precede or usher in the appearance of small flat spots, patches or blotches of a brownish or dark red-coloured efflorescence; they vary in size from the diameter of a pin's head to that of a pea or a three-penny piece. From these patches small, pimple-like bodies of a dark colour arise and project above the cuticle; in size and form they resemble small shot. These speedily enlarge into abruptly raised tubercles." Dr. J. M. H. McLeod, in his communication on the histo-pathology of yaws to the section of dermatology of the British Medical Association, in describing the histology of yaws squame thus writes: "The clinical appearance of the squame with its reddened, slightly raised and scaly surface was thus easily explained by the vascular dilatation, the deposition and infiltration of plasma cells and leucocytes, the epithelial proliferation, the interepithelial oedema, and the imperfect cornification."

Other authors beside those quoted have referred to this hyperæmia or engorgement of the capillary plexus of the skin at various points accompanied by desquamation, but the existence of such a squamo-erythematous eruption is denied by others besides Dr. Nicholls. Charlouis, for example, writes as follows on the subject: "According to some authors the patient presents, as the earliest eruption of frambæsia, red spots, upon which the papules afterwards develop. I have not observed such spots, and in my experience, the formation of an areola has only occurred when the papule had attained the size of a pin's head."

It is, of course, only on a sufficiently light-coloured

skin that these spots would be observable, but of their existence I have no doubt, though it is quite possible that they do not always occur. Shortly after reading Dr. Nicholls's remarks on the subject I saw such a squamo-erythematous eruption on a coloured yaws patient in St. Kitts.

I have described certain symptoms apparently due to yaws as forming a **third stage of the disease**, and exception has also been taken to the correctness of my views in this matter. These symptoms are **subcutaneous nodules**, which I termed "gummata," **ulceration of the fauces**, **destructive ulceration of the limbs**, **causing their contraction and atrophy**, **periostitis**, and **exfoliation of bone**. These symptoms Dr. Nicholls considers cannot be produced by yaws and must be due to either syphilis or tubercle. But he gives no other reason for this conclusion except the existence of the two last-named diseases in places in which yaws prevails.

If Dr. Nicholls could point out any symptoms indicative of syphilis or tubercle and of these affections only in those cases which he declares cannot be yaws, there would be some reason for his contention, but when in a case in which ulceration of the fauces has followed an attack of yaws and in which there is no evidence whatever of the previous or actual existence of syphilis, he attributes the lesion to the latter disease, his conclusion appears to me to be without foundation. His objection to a diagnosis of yaws in such a case because "there are no symptoms whatever concomitant with it to establish even the remotest relation to yaws" might be adduced with even greater force against the opinion that the symptoms are syphilitic. The patient at least had yaws before the ulceration appeared, though there were no concomitant symptoms of that disease, but there is nothing whatever to show that the patient ever had syphilis. Had the ulceration of the fauces followed an attack of syphilis, it would have been unhesitatingly attributed to syphilis, regardless of the absence of concomitant and confirmatory symptoms, but as it followed an attack of yaws it cannot be due to yaws.

But in denying that ulceration of the fauces is one of the later manifestations of yaws, Dr. Nicholls is contradicting others besides myself. Maxwell (1839) mentions excrescences in the throats of yaws patients which he considered were "not inappropriately likened to a piece of toasted cheese," and states that he has seen "the palate and fauces occupied with such excrescences when the dermoid tissue was very partially affected." "Occasionally a deep, excavated, yawy ulcer appears on the uvula or palate or back part of the fauces, sometimes at an early period of the disease, but more commonly at an advanced stage."

Dr. Imray, in Gavin Milroy's Report (1873), page 74, thus describes this affection of the fauces: "Although yaws break out more frequently about the face and forehead than perhaps on any other part of the body, yet it is rare indeed that the throat, palate, or nasal bones become affected. These parts may sometimes be involved when the malady has lasted for many months or years and the ulceration has become general; but such a consequence is by no means frequent." Imray also refers to the destructive

ulcerations affecting the bones which occur in yaws patients, "the specific nature of the ulcerations being indicated by the characteristic yaws showing itself here and there."

It is evident, therefore, that Maxwell and Imray—the former in 1839 and the latter in 1873—were not of the same opinion as Dr. Nicholls when the latter writes as follows, on page 343 of his report of 1893: "Mr. Numa Rat, therefore, in asserting that these symptoms are due to yaws, is only reviving old theories that were practically disposed of by authors at the beginning of the century."

Corroborative evidence of the occurrence of ulceration of the fauces, &c., as symptoms of yaws is supplied by Daniels, who thus writes in his Government Report on yaws, as observed by him in Fiji: "There are a series of pseudo-syphilitic phenomena met with in the natives, thought by some to have a connection with yaws. Syphilis is unknown among the natives."

"First among these is a destructive ulceration of the soft palate and fauces, and sometimes of the nose. With or without this there may be a destructive ulceration of the nasal cartilages resembling lupus exedens. Occasionally, either on the face or elsewhere, is a cutaneous affection resembling lupus vulgaris."

"I have twice seen this ulceration under 10, and it is common about 20. In rarer cases it occurs late in life, and in one woman, about 60, on whom I made a post-mortem, the larynx was involved; there were no tubercles in any of the organs, and neither gummata nor other signs of syphilis were present."

Professor Breda, Director of the Institute for Dermatology and Syphilis in the Royal University of Padua, in his "Contribution to the Study of the Brazilian Framboesia" (1895), has described an ulceration of the soft palate, fauces and larynx which he has observed in Italians who have returned to their country from Brazil, where they some years previously contracted the disease.

The statement that yaws is not an ulcerative affection is true as the same is true of syphilis. But no one would deny that ulceration occurs in syphilis because it is not generally observed during its secondary stage. Syphilitic secondary eruptions may ulcerate, but ulceration is so much rarer in the secondary than in the tertiary stage, that formerly all ulcerative processes were ranked as tertiary. Similarly we find that the secondary eruptions of yaws do not generally ulcerate, but that they may ulcerate and that ulcerations occur during the later periods of the disease there can be no doubt whatever.

It need not be pointed out that the tertiary symptoms of syphilis are not the result of the persistent activity of the virus in the system, but are changes due to the condition in which the tissues have been left by the previous influence on them of a specific poison whose virulence has been expended. This condition is a cachexia, and those who attribute to cachectic diseases the symptoms which I have grouped to form the third stage of yaws might with equal force maintain that the tertiary symptoms considered as being caused by syphilis are due to cachectic diseases also. And this did occur in former times;

and even in the early part of the last century, and still later the tertiary symptoms of syphilis and those due to retarded hereditary syphilis were often assigned to leprosy.

There is nothing in the nature of yaws to lead us to infer that it is incapable of producing the symptoms attributed to it in its latest stage. On the contrary, it would be strange if a disease which bears so close a resemblance to syphilis in the earlier portions of its course should not continue to show the same resemblance to it towards its close. It would be surprising if a general disease like yaws prevailing among ill-fed and uncleanly people, and affecting the system for years, did not eventually induce a cachexia like that of syphilis.

YAWS AND SYPHILIS.

The question whether yaws is syphilis modified by race and climate has been revived by Mr. Hutchinson's writings on the disease, and has given rise to much controversy during the past ten or more years. There are, however, two other questions which may be asked in this connection, and these are: Is syphilis yaws modified by race and climate? and, are yaws and syphilis modifications of a third disease? The proposition that yaws is syphilis modified by race and climate implies that syphilis originated in a non-tropical climate; but the suggestion which I was the first to make, that syphilis is a modified form of yaws would accord with the more general view that syphilis passed from the tropics to northern latitudes. I will limit myself, however, to a consideration of a fact which has been adduced in support of the contention that yaws and syphilis are, as they now exist, different diseases.

An apparently very effective demonstration that yaws and syphilis are different diseases has been given by Charlouis, who reported in 1881 that he had successfully inoculated a yaws patient with syphilis. This experiment and cases related by him and Powell, in which two men while suffering from yaws contracted syphilis, have been referred to very recently as very clearly showing the distinction between the two diseases. If such an experiment implied that two constitutional or general diseases had been found to exist concomitantly in the same patient, we should only need a few more examples of the same kind to settle the yaws-syphilis question. But the significance of this experiment, and the cases mentioned above in connection with it, is materially lessened when they are considered in connection with the opinions of those who hold, like Hirsch, that yaws is a purely local affection, and, like Nicholls, that it is sometimes a local, and sometimes a constitutional disease. If yaws exists in a patient as a local disease, as merely a skin affection, then the development of syphilis in such a patient would not be stranger than the appearance of syphilis in a person suffering from any skin disease. Similarly, from this point of view, when yaws is acquired by anyone affected with syphilis, we have the co-existence of a local with a general disease.¹

¹ Dr. Nicholls sees an analogy in this respect between yaws and tuberculosis. We may hence infer that as there are instances, though rare, in which a tuberculous skin disease has

But even when yaws and syphilis are concomitant as constitutional diseases, the stage of the one first acquired should be taken into consideration in estimating the value of such cases as those above mentioned. We do not know at what period of its course the virus of yaws as a constitutional disease loses its activity. The patient (Kamono) who was inoculated with syphilis by Charlois had been suffering from yaws during four years before the date of the experiment. Evidently, therefore, sufficient time had elapsed to render it probable that the yaws virus, if the disease had been a general one, had ceased to exert its influence on the system generally, and that its action was then limited simply to the cutaneous tissues. It is true that the patient had been inoculated a second and a third time with yaws, first with the softened crust and blood from a fungous tumour growing in his own skin, and next with the blood of another person suffering from yaws. Both the inoculations were said to have been successful; but the only symptom given in support of this assertion was the development of an ulcer in the first case at the site of inoculation, and the appearance of fresh tubercles whilst others were disappearing; and in the second, the development of a definite fungating growth with the characteristic crust at the point of entrance of the virus.

It is plain that in neither of these experiments can it be definitely asserted that yaws was successfully inoculated as a general disease. The occurrence of a fungating growth at the site of inoculation cannot possibly be considered sufficient evidence of the development of a general affection, and the appearance of fresh tubercles whilst others were disappearing might have been caused by the yaws contracted four years previously.

To be convincing, a similar experiment must be performed under conditions which leave no doubt as to the constitutional nature of the diseases inoculated. A person must be first inoculated with yaws and definite constitutional symptoms must manifest themselves before the inoculation with syphilis or *vice versa*, and also the second inoculation must be performed within such time after the first as will ensure the existence of the virus of the latter in the system in an active form.

I will close this paper by considering the **differences** which appear to me to exist **between yaws and syphilis**. The following are the most important of these differences:—

- (1) Yaws is limited to the tropics.
- (2) It may be either a general or a local disease.
- (3) Its micro-organism may exist in the soil.
- (4) It is not hereditary.
- (5) It is rarely contracted by sexual intercourse.
- (6) It is rarely observed in infancy.
- (7) It is commonly met with between the end of infancy and the tenth year of life, as many as 51 per cent. of the cases occurring during that period.
- (8) It is only about 8 per cent. of the cases that

occur between the twentieth and thirtieth years of life.

(9) Its primary lesion is a soft ulcer and never an indurated chancre.

(10) Its primary lesion appears two weeks after inoculation.

(11) Its secondary eruption does not show itself until about ten weeks after the appearance of the primary lesion.

(12) Its secondary eruption is not symmetrical.

(13) Its secondary eruption is monomorphous, the squame, papule and tubercle being stages in the development of the characteristic fungating growth.

(14) Its secondary eruption is characteristically fungating.

(15) Its secondary stage is never attended by lesions of the mucous membrane of the fauces.

(16) It never produces any eye affections.

(17) It never invades the viscera or the nervous system.

(18) It attacks bones not from within but from without by extension of the inflammation excited in the neighbouring cutaneous tissues.

(19) It presents the following histo-pathological differences from syphilis: *Positive*—(a) Greater extravasation of polynuclear leucocytes; (b) marked hyperkeratosis; (c) more pronounced proliferative changes in epithelium (except as regards condylomata). *Negative*—(a) Infiltration of plasma cells less dense; (b) linear arrangement of these rarely noted; (c) giant cells and chorisplaques never observed; (d) no fibrillation of the plasmomata; (e) no transitional branching connective tissue cells; (f) collagen less resistant and never organised; (g) no intracellular hyaline degeneration or colloidal degeneration of the fibrous stroma; (h) marked thickening or endothelial proliferation of the vessel walls not occurring to any extent.

The chief histo-pathological differences between yaws and syphilis, besides hyperkeratosis, are the absence of fibrillation in the growths of the former, which I pointed out in my essay in 1891, and again in my paper on yaws, contributed to the International Congress of Dermatology of 1896 (page 327), and of any intracellular hyaline degeneration or colloidal degeneration of the fibrous stroma.

The above differences are taken from the contribution to the histo-pathology of yaws, by Dr. J. M. H. MacLeod, already referred to. His study of the histology of the disease is exhaustive, and should be highly acceptable to those who are interested in the subject. He expresses the following opinions with regard to yaws and syphilis: "The differentiation of the yaws granuloma from the granuloma of syphilis requires even greater attention to detail than is necessary in the case of the other granulomata which have been maintained, and sections may be found, though they are, in my experience, the exception, from which it would have been impossible to venture more than a tentative opinion regarding the diagnosis. . . .

"Though the histological differentiation may be extremely difficult and at times practically impossible, still in sections in which the peculiar characteristics of yaws are well marked, it should be no more difficult to differentiate yaws from syphilis histologically

occurred in a patient, the subject of general tuberculosis, and *vice versa*, yaws may exist simultaneously in the same patient as a local as well as a general disease.

than it may be to distinguish between the histological pictures of tuberculosis and syphilis."

After a perusal of Dr. MacLeod's elaborate and most instructive description of the histological appearances presented by the plasmomata of yaws, one cannot but feel that, though he has pointed out several differences as regards the proportion and relative distribution of the elements which constitute the granulomata of yaws and syphilis, respectively, his account of the structure of the frambœsial plasmona has emphasised the resemblance already observed between the two diseases.

Correspondence.

To the Editor of the JOURNAL OF TROPICAL MEDICINE.

SIR,—As a constant reader of the JOURNAL OF TROPICAL MEDICINE, allow me to express my surprise at the terms in which you describe the recent action of the Society for the Suppression of the Opium Trade. To note only one expression, which may be taken as including all the rest, you describe the Society's action as "one, in fact, in which Government officials are flouted as consummate liars." Surely, Mr. Editor, you cannot be justified in this description? You are at perfect liberty to dispute the position which the Society has assumed in calling attention to a notorious fact. That fact is, that whereas the late Mr. W. H. Smith and Mr. Gladstone, both of them in their position as leaders of the House of Commons, distinctly stated that British Government policy was one of gradual restriction of the growth of the poppy in India, the actual condition of things, twelve years after Mr. W. H. Smith's statement, is that a larger area than ever is under poppy cultivation. You may agree with Lord Geo. Hamilton that he "cannot admit that there is any pledge of this kind which is operative at the present time or binding on the Government," but surely we do not charge either him or other Government officials with being "consummate liars," when it is urged that the policy of "*this present time*" is in direct contradiction to the avowed policy of ten years ago. The fact that Lord Geo. Hamilton has to use such language with regard to previous Government pledges as that they are not operative or binding on *the present Government* is proof that the Anti-Opium Society has good ground for its action, and that there is ample room for two opinions regarding these pledges. The Society has given no ground for any fair use of such language as you apply to it.

As to the subject itself, it is quite true, as you put it, that there are two questions, the one the relation of England and its Government to the opium trade, and the other, "the deleterious influence opium causes in China." But while you aver that these two questions have no direct or indirect bearing on each other, we assert the exact opposite. If cause and effect have no direct or individual bearing on each other, then England's opium policy and the opium misery of China have no relation. We assert that there is no single factor in the history of the last hundred years which has so powerfully contributed to the present opium misery of China as England's action (1) in the long and painful story of the opium smuggling traffic which led to the war of 1839-40; (2) in her continued refusal, after her victory, to bring the smuggling traffic to an end; and (3) in her refusal, after the legislation of the trade, to listen to the repeated entreaties of the Chinese Government to enter on some course which would bring the Indian opium trade to a close, a refusal which, in the counsels of Chinese despair, led to the awful spread of the poppy growth in China itself.

And we assert further, and emphatically, that if England

is to put herself in anything like righteous relations with China, and do something, instead of talking about it, which shall help China to reform, then her first step will be to bring to a close the Indo-Chinese opium trade. I do not like to follow you, Mr. Editor, in the use of hard speeches, but if there is one thing in our country's history which deserves the name of "indescribable meanness" it is her opium policy towards China.

49, Highbury Park, N. JAMES L. MAXWELL, M.D.
June 10th, 1902.

Current Literature.

PLAGUE.

PREVALENCE OF THE DISEASE.

INDIA.—During the weeks ending May 24th and May 31st the number of deaths from plague throughout India numbered 5,498 and 4,214 respectively. The decrease is marked, but the figures remain a long way above the corresponding period of last year, when the weekly return gave 1,658 only.

EGYPT.—During the weeks ending June 1st and 8th the fresh cases of plague in Egypt were stated as 14 and 4 respectively; and the deaths from plague during the same periods were 6 and 2 respectively. On the 8th of June 16 cases remained under treatment.

CAPE OF GOOD HOPE.—During the three weeks ending May 17th, 24th, and 31st the fresh cases of plague in Port Elizabeth, where alone the disease occurred, were 2, 1, and 0 respectively. During the same weeks the deaths from plague were 2, 0, and 0 respectively. Since the commencement of the outbreak 897 cases of plague have occurred in Cape Colony, of which number 431 died of the disease.

HONG KONG.—During the three weeks ending June 7th, 14th, and 21st, the reported cases of plague in Hong Kong numbered 53, 39, and 49, and the deaths from the disease 52, 39, and 51 respectively.

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- 2.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.
- 3.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.
- 4.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.
- 5.—Correspondents should look for replies under the heading "Answers to Correspondents."

The Journal of Tropical Medicine.

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Original Communications.

THREE LECTURES ON BILHARZIA,

Delivered at Kasr-el-Ainy Hospital, Cairo.

By FRANK MILTON, M.R.C.S.

Surgeon to the Hospital.

LECTURE III.

TREATMENT.

GENTLEMEN,—We have now come to the most interesting, although at present the most unsatisfactory, part of our subject, namely, the treatment of bilharzia under its various manifestations; the subject is unsatisfactory, because up to now we have got no means of attacking the cause of the disease and really curing the patient; but it is also most interesting, for the means may yet be found by which the patient may be rid of his unwelcome guest; and I can guarantee a fortune waiting for any one of you who can find means of killing the bilharzia worms in the veins of a human being without danger or risk to the host. Although as yet we are unable to radically cure the disease, still there are many things which we can do to mitigate the severity of its symptoms, and even in some cases to effect a cure, for in cases in which the fresh supply of eggs has been cut off owing to the death of the parent worms, the changes induced by the eggs previously deposited in the tissues which Nature unaided is unable to remedy may often be overcome by art. It has been advocated by some practitioners that injections of various chemicals and antiseptics should be made into the tissues, or even directly into the veins of the portal system, in order to kill the parasite, but happily I do not think those who advocate this method have ever had the courage of their convictions sufficiently strong to carry out their own proposals, and we may lay it down as a fact that at present there are no means of directly attacking the parasites themselves. The most recent suggestion of

this sort has been the subcutaneous injection of a solution of quassin, a principle obtained from quassia, with the idea of its absorption by the blood and the exertion by it of a vermicide action on the parent bilharzia; but although this may well be a step in the direction of a radical cure of the disease, I have not so far heard of any great success having been obtained from its employment. Quassi itself does not appear to be a very powerful vermicide, it being used as are decoctions of green tea, salt water and other mild anthelmintics, chiefly to destroy thread-worms, and does not appear to have much effect upon any other form of parasite; and if it requires a large enema of a strong decoction of quassia to kill a few thread-worms, I doubt if 0.002 of one of its principles transmitted through the whole bulk of the human body would prove to be more than a passing annoyance to a colony of bilharzia worms. I think from what will be seen later of the effect of the extract of male fern on bilharzial hæmaturia, that possibly subcutaneous injections of its active principle, filicic acid, would hold out more prospect of success, but I do not know that this has ever been tried.

All attempts at treatment, then, must resolve themselves into treatment of symptoms, and we will therefore deal with the symptoms according to locality, taking the organs in the same order as before, beginning with the bladder. For the early hæmaturia many methods of treatment have been advocated, especially injections into the bladder of astringent solutions, such as solutions of silver, zinc, and copper salts, and most of the solutions have been vaunted at one time or another as being certain of success; but before accepting these assurances of a beneficial result we must remember two things, first, that the hæmorrhage due to bilharzia is not always continuous, and it fairly frequently ceases for a time without any apparent cause; and secondly, that although the injections may control the hæmorrhage during their application, they cannot possibly cure it, as they are unable to do away with the immediate cause, unless they were

employed strong enough to exert sufficient influence on the mucous membrane as to alter its condition radically, which would be replacing one abnormal condition by another and probably a worse one. There is no doubt that relief may be afforded to the patient by washing out the bladder with mild antiseptics, such as boracic acid or boro-glyceride, but this will be due to the soothing action of the solution on the irritated bladder coats, and not to arrest of the disease. There is one preparation which, after long trial, I do really think can be relied upon to control the hæmorrhage, and whose effects would seem to be rather more than temporary and passing, although how it acts I am quite unable to say, and that is the liquid extract of male fern given in doses of 1 gramme three times a day. Over and over again in cases of simple bilharzial hæmaturia I have seen it act like a charm, controlling and abolishing the hæmorrhage within a day or two of its first administration, and relieving the bladder irritation at the same time. Being a powerful anthelmintic, and having so great and immediate an effect over a parasitic disease due to the presence of worms in the body, one would like to believe that it has the power to influence or destroy the parasite by being absorbed into the blood and acting directly upon the worm; but the very rapidity with which the results of the treatment are obtained is against this, for if the supply of eggs were suddenly to cease owing to the death of the parent worm, the changes set up by eggs already deposited in the tissues would still continue to give rise to the usual symptoms due to their presence until sufficient time had elapsed for them to be gradually got rid of in the ordinary way of Nature. It is therefore probable that the male fern acts in some way directly on the mucous membrane of the bladder and is merely a palliative agent and not a curative one.

As the disease advances the treatment consists more and more entirely in attempts to relieve the more urgent symptoms. The irritability and spasm of the bladder may be relieved to a certain extent by diuretic and emollient mixtures, and cystitis, which always occurs sooner or later, must be treated by washing out the bladder and the administration of urinary antiseptics, of which, in the majority of cases, salol in 0.75 gramme doses acts most readily and effectually. If calculi are formed they must, of course, be removed by crushing, and in this connection I think it may be laid down that bad cystitis amounting almost to disorganisation of the bladder is no bar to the performance of this operation, nor is it an indication for the performance of lithotomy rather than lithotritry, for I think the practice of this hospital proves that unless the kidneys are already the seat of bad secondary disease or even of pyæmic abscesses at the time of operation, all patients on whom a carefully conducted lithotritry is done will recover and be immensely benefited, but that cases of severe cystitis who are cut for stone are very liable to die, exhausted, without any attempt at healing of their wound having taken place. The relief given to these patients by the removal of their stones is very great, and that lithotritry is an especially favourable means of removing them is, I think, proved by a lately concluded series of 124 lithotritries which I have performed on patients with all degrees of cystitis without a single death, and

I am persuaded that by no other form of operation could these cases have been so successfully relieved.

The most difficult cases to know what to do with are the cases which come for treatment in the very late stages of the disease with disorganised bladders filled with bilharzial growth, and with constant passage of small quantities of foul urine with extreme suffering. The question in these cases is simply whether the bladder should be drained in order that the patient may be relieved of his constant tenesmus, or whether the patient shall be left to suffer unrelieved, for no treatment except drainage holds out any possibility of relief. If it were merely a question of making a perineal opening which was to remain patent for the rest of the patient's life, it would be a sufficiently serious thing to face, but one which in the majority of cases would be welcomed by the patient and which would be amply justified; but in reality it is much more than this, for it will be found that in nearly all cases in which the disease is far enough advanced to justify permanent drainage, the vitality of the growth filling the bladder is, owing to its own bulk and bad blood supply, so low that the mechanical injury necessary in introducing a large drainage tube and the irritation due to its retention in the bladder sets up a necrosis of the growth which sloughs and is then thrown off in masses. This extensive sloughing is necessarily a heavy drain on the strength of an individual whose resistance is already much broken down by his existing disease, and it will often prove too great to be borne and the patient will die, the operation being the direct cause of the shortening of a life miserable in the extreme, it is true, but still a life which the owner would not, even in his extremity, willingly give up. A certain proportion of the cases submitted to the operation of perineal drainage manage to survive the process of sloughing, and if they do this their condition after the loss of the mass of diseased tissue encumbering the bladder is very greatly improved and they pass the remainder of their life in a state of comparative comfort, but necessarily with an ever discharging urinary fistula in their perineum, for I do not think the drainage tract once fairly established will ever close, and it is certain that the remains of the bladder left after the sloughing of the whole of its diseased mucous membrane can never regain its functions as a contractile reservoir, but must remain a passive receptacle through which the urine passes. As far as I know there is no means by which it can be even approximately foretold if a given case will be capable of surviving the sloughing of the mucous membrane as represented by the mass of growth or whether the patient will succumb, for it will be found that some of the most miserable, broken-down patients, in whom the only apparent justification for the operation is to render death easier, will survive and regain their health and strength to a most extraordinary degree, whereas others in a comparatively fair state of general health will die rapidly as soon as the sloughing has fairly set in. Probably the result of the operation depends entirely upon the amount of damage already done to the patient's kidneys, but where the urine discharged from the bladder is represented by a quantity of stinking fluid, whose very amount it is impossible to measure with any accuracy, and whose

condition before its entry into the foul bladder it is impossible to ascertain, no certain indication can be obtained as to the state of these organs, unless, of course, they be found to be grossly hydro- or pyonephrotic, and even this condition which, where it exists, can be ascertained by direct examination is not so prejudicial to the patient as a kidney whose substance is riddled by minute chronic pyæmic abscesses, and which cannot be detected by palpation during life.

If drainage of the bladder is to be done at all, I very much prefer drainage through the perineum to drainage above the pubes. I see nothing to be gained by a suprapubic opening, and the patient is much more easily kept dry and comfortable if the opening is in the perineum; besides, the drainage is very much more efficient by the perineal route, the passages being near the lowest part of the bladder instead of at its highest. The operation I prefer is the old Cock's puncture through the perineum, with the introduction of two moderate-sized drainage tubes, whose introduction, by the way, is much assisted by oiling their ends, retained in place by a silk worm gut suture through each, and cut long so as to lie in a urinal between the patient's legs when he is in bed. The operation is extremely simple, and does away with the introduction of any unnecessary instruments into the bladder or down the diseased and probably tortuous urethra, and the opening into the bladder is the shortest possible, and in a direct line from the surface to the cavity to be drained. The double tube facilitates washing out, which is most necessary, not only for the purposes of cleanliness, and as far as possible disinfection, but also to keep the tubes from being blocked by blood-clots during the first day or two after the operation, and to remove sloughs later on. I generally retain the tubes about eight or ten days, after which period it will be found that their track will remain open and without much tendency to contract, and the opening will remain quite sufficiently free for the passage of the urine and of any sloughs that may still have to separate; of course, from the moment of coming under treatment the patient's strength must be kept up, and as far as possible increased by as much simple nourishment as he can by any means be induced to take and digest, and the most constant attention must be paid to the state of his dressings and bed to keep him free from the irritating urine, or sores will quickly develop in his badly-nourished skin.

The treatment of acute retention of urine when complicated with hæmorrhage is, necessarily, also by perineal section or Cock's puncture. In these cases the passage of a catheter is followed by no relief, for even with the employment of the largest instruments either no fluid can be drawn off, or if the urine does begin to escape the flow is soon arrested by the catheter becoming blocked with clot, and even repeated washings with hot water or weak antiseptic solutions, with the idea of breaking up the clot, will not give really satisfactory results, for it is almost, if not quite, impossible to evacuate the whole contents of the bladder in this way, and even if the attempt were successful and the bladder completely cleared, it is not at all sure that the hæmorrhage would not continue, or recommence, and the process of evacua-

tion have to be undertaken again. Whereas by an incision in the perineum the bladder can be thoroughly evacuated in the shortest possible time and with the least possible suffering to the patient, and should the hæmorrhage recur the part is entirely under control and everything in condition for immediate further treatment. In these cases I prefer to do a median perineal cystotomy on a staff, rather than a Cock's puncture, for the extra room given by the larger incision is of great help in the manipulations generally necessary in removing the clot. Having made your opening into the bladder, you should introduce your finger through the wound and thoroughly break up the clot, not being more rough than is absolutely necessary, for you must always remember that you are dealing with a badly diseased part and one which very easily sloughs, for these cases of hæmorrhage with retention only occur in advanced cases with an excess of growth. You will find that masses of growth readily come away, and it is very tempting to go on and try to clear out the whole of the diseased material; but this is a mistake, for although you may in this way remove large quantities of bilharzia matter, you cannot remove the whole, and the accompanying hæmorrhage although not excessive is of serious moment to the patient in his broken state of health. The thing to do, then, having once broken up the clot, is to introduce through the wound two full-sized lithotripsy evacuating catheters and wash through them alternately with a large quantity of saline solution, continuing to wash through until the water is returned clean, and then to fix in two moderate-sized drainage tubes as in the case of drainage of the bladder. It will generally be found that with a free exit for the urine the hæmorrhage stops, but should it recur, the best method is to again wash out the bladder through the tubes with a solution of creolin, either at a temperature of 40° to 45° C., or with the same solution iced.

The treatment of urethral urinary fistulæ due to bilharzia is, on the whole, more encouraging than the treatment of any other form of bilharzial infection, for in the majority of cases the patients can be cured, at least of their local disease, and the treatment is really of the simplest, and consists in the free and wide excision of the fistula and its surrounding tissues. Of course, in some of the most severe cases of fistula, when the patient's perineum and even nates and thighs are a mass of fistulous openings, this free and wide excision is impossible, and we must be content with more imperfect operations; but even in these cases, with patience and endurance on the part of the patient, and perseverance and ingenuity on the part of the surgeon, the worst cases may be greatly benefited and often cured. In the great majority of cases of urinary fistula it will be found that there is no stricture present, and this fact greatly increases the prospect of cure. In many cases where a stricture would seem to be present, and where a small-sized catheter is obstructed, a No. 12 English metal catheter may be passed with ease, the obstruction being due to distortion of the canal and not to stricture. In some cases true stricture will be found, and where it does exist cure is very difficult, both as regards the fistula and as regards the stricture, but even in these cases,

if everything else fails, much may be done to benefit the patient by substituting a direct, fairly healthy escape for the urine through the perineum in place of, as is usually the case in these patients, a number of unhealthy, badly-placed and tortuous fistulous openings. As we have seen, stricture only occurs in roof fistula as the result of very extensive infiltration and destruction of a large part of the urethra, and is therefore practically incurable, for the part of the urethra which has been destroyed is only represented by the ordinary granulation tissue entangled, as it were, in a fibrous network such as forms the lining of bilharzia fistulæ, and although an instrument may be easily forced through this tissue there is not sufficient coherence in the tissue itself to even permit the formation of a canal in it. As a rule, then, stricture accompanies floor fistula only, and is due to the alteration of the walls of the urethra by chronic inflammation, the result of the constant passage of the irritating discharges from the progressing fistula, and here also the change is so extensive and the alteration of the part so profound that these strictures are the most intractable with which we have to deal, for although a passage may be forced through the infiltrated and narrowed urethra, and the catheter maintained in position for a length of time, yet when the instrument is at last withdrawn the canal will for a certainty contract again immediately to its former dimension.

In a case of fistula or fistulæ in the perineum without stricture the method of operation is as follows: The patient, being anæsthetised, is held in the lithotomy position and a full-sized metal catheter is passed into the bladder; an area of skin is then marked out with the scalpel, including, if possible, all the fistulæ present, with a fair margin of skin around them. It will generally be found that the incisions have to be made extending to both sides of the middle line and often encroaching upon the scrotum above and coming near to the anus below. The incisions are then carried boldly down through the tissues, and gradually sloping somewhat towards the middle line until the level of the urethra is reached. The bleeding, which is free, is easily controlled by picking up the vessels as they are cut through, and no attention need be paid to anatomical details which have already been modified by the bilharzia infiltration. Having reached to about the level of the urethra, this canal is to be searched for and then carefully isolated and cleaned of infiltrated and thickened tissue which, if you find the right line of cleavage immediately on the urethra itself, is very easily accomplished. It will then be found that the granulating tract of the fistula, which up to this point should not have been seen as it should be hidden well in the centre of the block of tissue being removed, passes on one side of the urethra round to its upper surface. As this part of the tract cannot be cut away without doing serious damage to the surrounding parts, the operator has to content himself with thoroughly scraping it out with a sharp spoon until every visible vestige of granulation tissue has been removed. The larger vessels, which have been divided in the course of the operation, are then ligatured, and the wound plugged widely open with antiseptic gauze. In the perineum the whole wound

should undoubtedly be left open whatever its size, but if the wound has extended into the scrotum the skin in this, the upper part of the wound, may be sutured, provided always that wide access be left to the deepest parts of the wound leading to the exposed urethra. The wide open wound will naturally take longer to granulate up than if the sides were allowed to approximate to some extent, but it will be found that what is apparently lost in time will be more than regained in certainty of cure, for it must be remembered in the deepest part of the fistula, the removal having been by means of the sharp spoon and not by the knife, it is pretty certain some of the granulation tissue will have been left, and this and the roughly-treated healthy tissue in its neighbourhood will have to be thrown off by suppuration, which can only be done satisfactorily upon what is rendered as nearly as possible an open surface.

In Europe and in non-bilharzial countries generally urinary fistula is always due to stricture, either gonorrhœal or traumatic, and the routine method of treatment is to dilate the stricture, incise the fistula, and tie a catheter in the urethra until healing is well advanced. The same methods have been followed in treating fistula due to bilharzia in this country, especially as regards the tying-in of a catheter after operation, but I think this is a mistake, and I think the practice is not only needless but even harmful. The ends aimed at by those who tie in a catheter are two: first, to maintain or restore the calibre of the urethra, and second, to prevent the escape of urine through the wound and possible interference with the healing process owing to its irritative properties. Of course, where a dilatable stricture exists the practice is proper and necessary in order to provide a free exit for the urine other than by the fistula; but, as I have stated above, in the majority of cases of bilharzial fistula no stricture exists and so dilation is unnecessary, or where true stricture does exist it is by its nature so undilatable that its condition cannot be improved by the passage or even the retention of instruments, and as regards the maintenance of the calibre of the urethra after operation, the opening in the urethra, especially in roof fistula, is as a rule small and situated in the long axis of the canal, so that the amount of contraction due to its healing is altogether unimportant, and does not require special treatment for its prevention. Again, I do not think that any catheter tied into the urethra, however tightly it may be grasped, will drain away the whole of the urine after the first twenty-four hours, but in every case it will be found that a certain amount of urine will find its way along the side of the catheter and escape from the fistula if such exists; and again, provided the urine has a free and unobstructed outlet I do not think it will irritate even freshly-cut tissue. On the other hand, the presence of a rigid catheter in his urethra is intensely painful and very annoying to the patient; it causes him to maintain an extremely rigid and fatiguing position in bed, and it is very difficult to manage properly from the point of view of emptying the bladder, and after a very short time it will set up urethritis and probably increase the irritability of the already diseased bladder. Owing to these facts I never tie a catheter in after operating for fistula,

and I do not think I have ever seen a case where the advantage of keeping in an instrument would not have been outweighed by its very serious drawbacks.

In the unfortunate cases where the urethra has been completely destroyed in whole or in part the procedure to be adopted is to excise the fistulæ, which in these cases are usually multiple, in the thorough manner described above and to leave the healing process to go on until the wound has closed as far as it will, when there will remain a sound scar perforated about its centre by a fistulous opening, which will remain permanent and over which the patient will, as a rule, have a fair amount of control, seeing that its origin is in the urethra well in front of the prostate. No special means need be taken at the time of the operation to provide an epithelial-lined course for the new fistula, as it will be found that after a certain period there will be no further attempt at healing and the fistula will remain permanently open.

It will at times happen with very extensive disease and widely-distributed fistulæ that the operation will have to be repeated, occasionally several times over; but there are, on the whole, few cases that cannot eventually be cured, provided always that there is no bad stricture of the urethra; and still fewer that are not capable of great improvement, even in the presence of an obliterated urethra.

The most difficult cases of all to deal with are the cases where the opening in the urethra is in the usual position just in front of the bulb, but the disease, instead of making for the nearest skin surface, has travelled along the under-surface of the corpus cavernosum and has made its exit in front of the scrotum or on the pubis. Scraping, excising, and plugging, however thoroughly done along the course of the fistula, even in conjunction with a wide counter-opening in the perineum, will never succeed in curing these cases, and the sole method to be adopted is to boldly slit up the scrotum in its whole length, dividing it into two separate parts along the raphé, and to expose the whole length of the fistula, to plug the wound open and leave it to granulate up in its whole extent, not venturing even to approximate the two halves of the divided scrotum until the granulations have filled up the cavity well above the level of the urethra.

The treatment of floor fistula differs from that of roof fistula owing first, to the presence of stricture, and secondly, to the large amount of dense fibrous tissue between the surface and the urethra, rendering the definition of this canal very difficult. The treatment of the stricture itself need not detain us, for as a rule it will be found to be quite undilatable if it is of any standing, and owing to its extent it does not lend itself particularly well to any cutting operation, or rather, perhaps, the only cutting operation likely to be of any avail is that shortly to be described, as done at the time of operation for the accompanying fistula. Before considering the treatment of the true and complete floor fistula we have first to describe the treatment of the incomplete fistula whilst it is still in the form of a peri-urethral abscess. We described these abscesses in a former lecture as consisting of a dense mass of new fibrous tissue forming a tumour of stony hardness, and having in its centre a cavity containing stinking pus, and the object of your treatment

must be not merely to evacuate this pus, but also to remove the whole of the fibrous tissue enclosing it as well. To do this you must make a free incision in the middle line over the tumour through all the overlying tissues, into and through the whole extent of the tumour itself and into urethra, which you incise in its long axis through the whole extent of the tumour and clear beyond it at either end, this incision will pass through the opening of communication between abscess cavity and urethra, which will be bound to be of a fair size, lying in the long axis of the urethra and with ragged, granulating edges. It does not much matter whether you have introduced a catheter into the urethra or not before beginning your operation, for the urethra has to be incised, and incised freely, and owing to the density of the overlying structures a catheter is of very little assistance in defining the position of the urethra, the true guide to which is the cavity of the abscess and its internal opening. You are compelled to incise the urethra itself because in these cases it is altogether impossible, working from the outside, to clear away the new tissue, for you are unable, accurately, to define the position of the urethra, and this uncertainty restrains your freedom of action so that either fearing to damage the urethra you hesitate to remove enough of the new tissue, or else in making too free incisions you may easily find that you have also removed a slice of the urethra, which loss in the already narrowed state of the canal is irreparable; whereas, if you split the whole of the fibrous tissue and incise the urethra freely in the middle longitudinal line you have the whole canal immediately under your eye and can define its limits and dissect away the whole of the new tissue from within outwards without any difficulty or hesitation, and the incision in the urethra instead of being prejudicial to the patient, is rather of an advantage, as it not only readily heals without any extra narrowing, but also partakes more or less of the nature of an external urethrotomy and probably increases the calibre of the urethra at this point as the scar tissue between it and the surface contracts. The treatment of the complete fistula does not differ from the treatment of the bilharzial peri-urethral abscess, except that the part to be removed includes an elliptical area of skin round the mouth of fistula, but the definition of this area had better be left to the last stages of the operation, and the first incision be made as for peri-urethral abscess in middle line, for if you begin by making incisions on the skin surface to include your fistula you will find that you are tempted to continue deepening these incisions and are led on further than you proposed, until you find yourself involved in an attempt to excise your floor fistula from without inwards, instead of, as you should, from within outwards, and you soon get into a state of uncertainty and indecision, consequent upon not knowing exactly where you are in relation to the urethra; but if you ignore the presence of the fistulous opening and cut down through it straight into the urethra, all temptation is avoided and you can complete your operation from within outwards with perfect ease and certainty. Of course, the removal of all infiltrated tissue must be as complete as in the case of roof fistula, and the wound must be left equally wide open.

In those cases where the stricture holds out any prospect of cure this may be undertaken during the after-treatment of the fistula, that is, the stricture should be dilated as far as possible whilst the patient is still under chloroform for his operation and the dilatation kept up, or, if possible, increased, by the subsequent daily passage of metal sounds during the whole time of the healing of the wound and at longer and gradually increasing intervals afterwards.

A much more seldom met with form of urinary fistula is fistula connected immediately with the bladder, resulting from a penetration of the bladder disease through its muscular coat, causing adhesions between bladder and belly wall, and finally perforation and the formation of a fistula situated either between pubis and umbilicus or in one of the groins. These fistulae only occur in most extreme cases of the disease, when the whole bladder is disorganised and its cavity filled with bilharzial growth which prevents the escape of the urine by the urethra, and I believe they are altogether incurable. I have only had a few of these cases, some four or five, and in spite of wide excisions, even combined with suture, and a perineal opening, I have never managed to cure any of them. It may seem rather a rash and desperate proceeding to inflict such serious wounds as are implied in the description of these operations for fistula upon patients who are in a debilitated state from the presence of advanced bilharzial disease; but whether it is that they have acquired a habit of resistance from the sufferings they have borne so long, or whether their release from the worst of these sufferings improves their physical tone, their recuperative powers they exhibit after these mutilations are most surprising, and although I have removed large masses of tissue and inflicted cavernous wounds, I am happy to say I have never lost a case as a direct result of operation for urinary fistula.

The treatment of bilharzia of the vagina is, I am sorry to say, judging from the few cases I have been privileged to treat, at present most unsatisfactory, and the futility of such methods as douching and disinfecting and the application of all sorts of possible medicaments on this easily gettable mucous membrane only serves to indicate how little influence our necessarily more feeble efforts can have on the mucous membrane of the bladder when affected with the same disease. The only proceeding holding out a prospect of cure is excision of the thickened and infiltrated mucous membrane, and this, of course, can only be done where the disease is limited in extent. If the cases presented themselves in a sufficiently early stage, with the disease limited, as I believe it generally is in its early stages, to the posterior wall of the vagina, the operation would be simple, and probably, for the time being at any rate, effectual; but although I have done excision of a large extent of mucous membrane in two of my cases I was not altogether satisfied with the result, although the patients expressed themselves as benefited, and I think they were so to a certain extent.

The treatment of bilharzia of the rectum in its early stages consists in allaying the irritation of the part, and lessening, as far as possible, the hyperaemia and secretion of mucus. This can best be done by local

sedative and astringent applications, as enemata of starch and opium, or of solution of sulphate of copper, and undoubtedly much relief is given to the patients by this treatment; but the disease tends to advance in spite of anything we can do at present, and when it has arrived at the stage of formation of polypi its treatment is even more unsatisfactory than that of bilharzia of the vagina, for here, although it would seem to be a very simple thing to remove the polypi by excision or ligature, or even to excise the affected part of the gut, yet in practice it will almost invariably be found that the disease extends so far up the intestine that it is impossible to go high enough up to get to the healthy mucous membrane; any attempt at systematic ligaturing and removing by scissors, for instance, leading one on and on until the ligature can no longer be tied owing to the distance from the surface, the growths not even diminishing in number or size, but rather increasing, the higher one goes. In cases without prolapse, a certain amount of relief may be given temporarily by thoroughly stretching the sphincters, and at the same time removing any especially large polypi, but the relief is only quite temporary, and although the patient obtains a respite he invariably returns to his former condition. Latterly, I have been trying the effect of stretching or incising the sphincter, and then swabbing the gut out as high as I can reach with a pledged of cotton-wool held in a forceps, and soaked in a 1 in 10 solution of chloride of zinc, allowing the solution to remain in contact with the tissues for about one minute, then drying out the excess with dry cotton pledgets, and finally flushing out the gut with copious enemata of saline solution. This is followed by no unpleasant results, indeed the patients express themselves as being relieved; but I have not as yet done enough cases, or been able to follow them up long enough, to determine as to whether they have received any permanent benefit. In cases with prolapse due to bilharzia, it is rather difficult to decide whether excision of the prolapsed portion is justifiable or not. Of course, if the relief were likely to be permanent there would be no question as to the advisability of the operation in suitable cases, but the same reasons that make removal of polypi ineffectual renders the result of any operations for prolapse also uncertain. In some cases lineal cautery, with destruction of multiple areas of the whole mucous membrane, seems for a time to prevent the descent of the bowel, and in the majority of cases admitting of any form of operative treatment perhaps this is the best course to pursue, always remembering that great after-care is necessary to remove the sloughs which are cast off and to keep the rectum clear. This is best done by enemata of olive oil twice daily for the first three or four days after operation, and then by enemata of boracic acid solution twice daily until all the sloughs have come away. In cases still more favourable than these, and there are a certain number in which prolapse occurs fairly early in the disease, and when the patient's health is by no means broken, I think excision of the prolapse is not only justifiable but called for. These are, as a rule, cases of fairly well-nourished men whose forcible and continuous efforts at expulsion of the irritation felt in the rectum have caused a prolapse,

as it were, more traumatic in nature than the majority of cases, and when the suffering is more acute and the prolapse with difficulty reduced, owing to the amount of contractile force still remaining to the sphincter. These cases, I think, can be relieved for a comparatively long time by the operation of excision, and in them I have done it with some success, although, unfortunately, the cases have been few. The important things to remember in operating on these cases are two: first of all the absolute necessity of preserving both sphincters intact, and secondly, to divide the gut as high up as possible. My method of operating is as follows: Having the patient under chloroform and in the lithotomy position, I pull the prolapse down as far as it will come and attach two pairs of artery forceps to its apex when protruded, one in the middle line in front and the other in the middle line behind; I then search for the upper edge of the internal sphincter, or if this is impossible to find owing to the thickening of the mucous membrane, I allow for its breadth about an inch and a half above the junction of the mucous membrane and skin, and cut through the gut at this point in the middle line anteriorly: in some of these cases it would seem that the peritoneum comes lower down than normal, for in about half the cases operated upon the peritoneal cavity has been opened by this incision and this very much simplifies the operation; if the peritoneum be not immediately opened a way must be made either with the finger or with the handle of the knife through the connective tissue between the rectum and bladder until the peritoneum is reached and opened. Having opened the peritoneum the incision begun in front at the edge of the internal sphincter is carried round the circumference of the bowel, the edge of the sphincter being picked up at intervals of about an inch with artery forceps, in order to keep it under control. The incision, starting from in front, very soon leaves the peritoneum, as this does not come nearly so far down on the rectum behind, but the limits of the gut are very easily defined and the division presents no difficulty. The lower end of the intestine having been freed the prolapse is pulled down as far as it will come without exerting too great force and the gut is again cut through in front and the upper cut edge is caught with forceps. A silk-worm gut stitch is then passed from the inside of the upper gut through all its thickness, made to traverse the peritoneal cavity and passed through the sphincter from its peritoneal side into the lumen of the bowel and clamped in position with forceps. The incision in the upper end of the gut is then continued round its circumference, the edge being clamped and a stitch being introduced at about every inch as it is cut. When the circuit is complete the upper end of the intestine will be completely controlled by the forceps clamped on it and all the sutures will be in place. All the vessels which have been divided and clamped during the double division of the bowel and which are somewhat numerous behind are now secured by ligatures, and nothing is left at the seat of operation but a double row of clamping forceps and the sutures in place, the upper end of the gut being prevented from escaping upwards into the peritoneal cavity by its attached forceps. Some bleeding points will be found in the mucous membrane itself and

which are difficult to secure, as the thickened tissues break away on being caught with forceps, but these do not matter, as all hæmorrhage from them will be stopped by the sutures when they come to be tied. All that remains to be done now is to tie off the sutures, and this is done beginning from the middle line in front and tying them in order, one on each side of the gut, until the circuit is complete, the nearest forceps being removed as each stitch is tied. The sutures are kept long until the whole of them are tied in order, that they may take the place of the forceps in maintaining the control of the gut. When the sutures are all tied the wound is gone carefully over again, and any accessory suturing that may be necessary to accurately approximate the divided edges of the gut is done; and finally, when all is quite secure, the sutures are all cut short. As soon as the sutures are cut the gut shoots back into the abdomen by its own contraction, this being brought about by the intestine having been forcibly brought down and divided about the true line of protrusion, and with it carries part of the anus, including the internal sphincter, this muscle being drawn back into its natural position. The part is left altogether undisturbed, and after about eight or ten days the sutures which have not cut their own way out should be removed, and this will be found a matter of some difficulty, owing to the amount of contraction which the sphincter will be found to have regained. The operation is one of some severity, and should only be undertaken in favourable cases, but in those cases when it can be fully carried out the result will be found most satisfactory, this good result depending upon the fact that the sphincters are not interfered with, and that they are therefore in a condition to regain their functions.

In cases of bilharzial infiltration occurring in the skin, the treatment simply consists either in complete excision or a thorough destruction, by scraping of the whole of the granulation tissue, followed by the simplest form of antiseptic dressing; and although the cicatrization of the wound may be tedious owing to its extent, the cure will in the end be complete.

Bilharzial affections of other organs have not yet come into the range of practical surgery, although we may hope in the future to extend our operations as far and as fast as our knowledge of the different forms of the disease increases; for I am sure that as we search more carefully and closely for extensions of the disease, such extensions, at present unsuspected, will be found; but however widely we extend our search and our treatment the disease will never be capable of true cure until we find some method of attacking the parent worms in their at present inaccessible habitation, and I sincerely trust that this signal service to the Egyptian race may be achieved by a native Egyptian surgeon educated at Kasr-el-Ainy.

MYIASIS — THE LUCILIA MACELLARIA — THE SCREW WORM.

By JOHN W. LINDSAY, M.A., M.B., Ch.B.(Aberd.)
Villa Concepcion, Paraguay, South America.

CASES of Myiasis are very common in this country. The screw-worm (*Lucilia macellaria*) is found embedded in the skin in all conceivable situations, in the legs, arms, chest, back, face, and scalp.

It is very seldom that all the animals about a farm are perfectly free from this pest; hence it is that the patients who come for treatment are generally of the farming class. Many of these have no adequate means of treating themselves, and it sometimes happens that very bad sores result. Unless one is familiar with the condition, mistakes in diagnosis are apt to be made.

In one case of a small tumour of the scalp I could detect no opening or sinus, and considered it a sebaceous cyst. Later, when it came to be treated, it was found to contain a screw-worm. A lady had one in her upper arm. She had felt no symptoms to call her attention to the part until one day she noticed the swelling, and to her alarm saw protruding from the opening at the apex of the tumour the head of the larva. She used to sit and watch it, and her remarks were most amusing. "Oh, the horrid looking beast! he's gone again! I go to catch him, and just when I think I have him he disappears!"

A boy of 16 consulted me in regard to a swelling in his right eyelid. He had felt the first symptoms about three weeks before. On waking one morning he felt a slight itching of the margin of the eyelid, and noticed a slight localised swelling. Since then it had increased in size until now it was as large as a hazel nut. The patient stated that it had burst several times, and that a dirty-looking bloody discharge had escaped.

At times he suffered excruciating pain, and for several nights before he consulted me he had got no sleep. As I had no means of treatment with me and no antiseptics, I merely gave the swelling a very superficial examination, and diagnosing it as a Meibomian cyst arranged to operate the following day. I washed out the eye with weak sublimate lotion; the result was rather disconcerting. The patient drew himself together, and groaned with pain.

I hastily introduced some solution of cocaine. The effect this time was rather surprising, for from a small sinus just above the line of and slightly hid by the eyelashes there shot out the head of a maggot!

On evertting the lid I found that the larva had burst through the palpebral conjunctiva, and was lying up against the fornix and bulb.

When removed it was found to be fully three-quarters of an inch long, and about three-eighths of an inch in diameter at its thickest part. It had the typical slightly irregular rings or segments, armed with minute dark-brown horny spines. This explained the attacks of severe pain from which the patient suffered, for every time the larva moved or was disturbed it must have caused great irritation of the conjunctiva.

I touched the sinus or opening with pure carbolic, and applied a simple bandage and dressing.

Three days later the eyelid looked almost normal.

The boy stated that once before he had had one in the skin of his chest. He had noticed them in the dogs and other animals about his home.

The local remedies for "ura," as it is called, are tobacco ash or juice, or kerosene. The presence of the abraded surface caused by the larva is frequently the predisposing cause of what is locally known as "espasmodura," a kind of diffuse cellulitis, usually of the lower extremities.

HARROGATE AS A HEALTH RESORT FOR TROPICAL INVALIDS.

By P. A. NIGHTINGALE, M.D., EDIN.
Formerly of Bangkok, Siam.

THE number of persons who have resided any length of time in the tropics and who have not suffered from some ailment incidental to the climate is but small, while the majority of those who have escaped, on returning to a temperate zone soon feel that they are not "quite up to the mark," and wisely seek advice as to what they should do, and where they should do it.

In these latter an examination quickly shows either that they are anæmic, or that their livers have had too much work thrown on them, the skin no longer being bathed in the perspiration it has been accustomed to for years, or that their spleens are a little enlarged, or their digestive organs cannot assimilate the home food to which they have been so long strangers.

It is borne in on them that life in London, or any other large town, with its round of gaieties and dinners, want of regular exercise and air deficient in ozone, but makes them worse, and they see before them a chance of having to go on the sick list and all enjoyment of their well-earned holiday lost.

A visit to one of the South Coast seaside watering places is tried, usually on account of its mildness, but found wanting—for to the tropically-anæmic person it brings neither sound sleep, healthy appetite, nor removal of that muscular slackness of which he complains so much.

The secret of his troubles lies in the fact, which practical experience alone teaches, that the tropical resident requires bracing hill air to invigorate and refresh him, and that without it he will not derive the full benefit of his trip home.

To such a person Harrogate is the ideal resort during the summer months, and a stay of a few weeks in it will give him a new lease of life.

Situated some 400 feet above sea-level, on the highest table-land between the Irish and North Seas, and almost equi-distant between the two, its air well charged with oxygen and ozone, Harrogate (in the West Riding of Yorkshire) during the last few years, has become one of the best known and most patronised inland watering-places of the kingdom.

Mr. Paul, Borough Meteorologist, has recorded the following observations during the year 1901:—

| Month. | Mean Temp. of Month. | Mean Relative Humidity of Air. | Rainfall. |
|------------------|----------------------|--------------------------------|-----------|
| May | 50.2 | 73 per cent. | 0.78 ins. |
| June | 55.2 | 68 " | 1.48 " |
| July | 63.4 | 79 " | 1.44 " |
| August | 59.5 | 80 " | 1.38 " |
| September | 55.0 | 85 " | 1.00 " |
| October | 47.4 | 91 " | 1.89 " |

The rainfall for the whole year was 24.36 ins., and the average during the previous eighteen years was 29.25 ins.

The mean reading of the barometer at sea-level and zero was 29.897, and the average mean for eight years was 29.902.

The amount of sunshine recorded was 2171 hours 34 minutes, while the death-rate during the last seven years averaged 12.3 per 1000.

The town is built on a series of gently-undulating slopes prettily laid out in gardens, admitting of the graduated exercise in the Nauheim treatment of cardiac disease being carried out to perfection.

The baths and waters are, however, the main attraction of Harrogate, and few places have been so favoured by Nature and a wise administration as this fashionable resort.

The waters naturally divide themselves into two main classes—the sulphur, in the form of sodium sulphide, and the chalybeate, in the form of ferrous carbonate. Thus we have the strong and mild sulphur, the saline chalybeate and the pure chalybeate springs in numerous varieties and strengths.

The well-known action of sulphur in liver and other affections need not be touched upon here, but the Harrogate waters also contain an appreciable amount of the sulphate or carbonate of barium which, as a cardiac stimulant, is a valuable adjunct, especially when the waters are taken for their alterative action.

Among the saline chalybeate group, one especially, the Kissingen Spa (so called from its close resemblance to the Kakocz spring of Kissingen, in Bavaria) deserves mention, for its great value in the treatment of anæmia and chlorosis.

Harrogate now boasts of the finest collection of baths in Europe, there being over thirty different kinds of baths, douches, and massage; among these must be mentioned the d'Arsonval high-frequency electric currents; the Dowsing radiant heat and light treatment; the Greville hot-air treatment; the carbonic acid bath (Nauheim); the liver pack and the electric sulphur bath. The Aix-la-Chapelle inunction methods for syphilitics are also carried out with great success by skilled attendants, while dry and wet massage in every form is made a speciality of. Thus the tropical invalid will find waters and baths of every variety to suit his ailment, while the fine bracing air, inducements for outdoor exercise, and general gaiety during the season, will complete the "cure" of those who only need a general bracing up of the nervous and muscular systems.

Finally, it should be noted that after the end of October Harrogate is rather too cold for the tropical resident, who might then, with benefit, go to a more southern watering-place, such as Bath.

OBSERVATIONS ON PLAGUE.

By PROFESSOR C. TERNI.

Of the Bacteriological Institute of Messina, Italy.

THE diagnosis of plague presents very considerable difficulties, especially during the initial outbreak of the disease. This is not due to ignorance nor to any wish to conceal the disease, but is owing to the want of positive clinical and pathological features at the early stages of the outbreak. In consequence of this uncertainty such terms as lymphatitis, pernicious lymphatitis, malarial lymphatitis, &c., are bestowed upon these cases, thereby causing, not infrequently, serious consequences, owing to initial cases of plague remaining undiagnosed and becoming diffused foci of infection.

MILD CASES OF PLAGUE.

In every country in which plague has recently appeared benign cases of the disease have been described; some of these cases end in spontaneous cure, inducing many observers to attempt to establish differences between the mild and the severe forms of plague, which occur after the outbreak has lasted some time.

In Egypt, for example, long discussions took place on this subject, and the initial cases of mild plague were diagnosed as lymphatitis, or lymphadenitis, and it was stated that such acute affections were common every year during the hot weather, and especially during the fall of the Nile. In India, at Oporto, and especially in Brazil, the same opinions prevailed, and in all these countries it was subsequently demonstrated that the so-called cases of climatic adenitis were really cases of true plague.

It is easy to understand the importance of obtaining some ready means of diagnosing between plague and ailments which, whilst simulating it in many respects, may, or may not, belong to the same etiological factor. So long as there is any confusion between plague and allied disorders there is a great danger that the earliest cases of the disease escape detection, and all subsequent attempts at stamping it out are rendered much more difficult, or altogether impossible. Having had the opportunity of studying over 1,000 cases of plague, mostly at the Paula Candido Hospital, in Rio-de-Janeiro, I propose to recount some of the characteristic features of the disease and of the differential characteristics of the disease.

DIFFERENTIAL DIAGNOSIS BETWEEN LYMPHATITIS AND BUBONIC PLAGUE.

The initial cases of plague are almost invariably bubonic in character, and it is only when the disease has lasted some time and the plague bacilli have acquired a marked degree of virulence that gastrointestinal, septicæmic or pneumonic forms appear.

Brazilian physicians have investigated closely so-called pernicious and malarial "lymphatitis" under which appellation they classed together all cases of adenitis and lymphangitis which ran a rapidly infecting course. These, however, for the most part present the usual symptoms of phlegmon in temperate climates.

The affection usually commences with a phlyctene, papule or cutaneous inflammation, the result of slight injury or the bite of an insect. At the seat of infection a slight necrosis or eschar forms, similar to a malignant ulceration (carbuncle) with livid margins, followed by hyperæmia and œdema of adjacent tissues. Subsequently lymphangitis and adenitis supervene, accompanied by polyadenitis. Fever with rigors suggest a malarial type, but quite unjustifiably, and as Rho remarks, inflammatory and phlegmonous complications, in persons rendered anæmic and depressed by long residence in a hot climate, are likely to arise without finding malaria as a determining cause. In all the cases I examined I found septicæmia, due to the presence of streptococcus pyogenes, in the blood; very occasionally staphylococci were met with and in two cases diplococci; the presence of these cocci excluded the possibility of the ailment being due to malaria.

In tropical countries other affections, such as carbuncles and malignant œdema, give rise to lymphangitis and adenitis, but modern methods of investigation differentiate these nowadays.

In plague, the term lymphatitidis, which is meant to include lymphangitis and adenitis, does not apply, as inflammatory tracts along the lymphatics is not the rule. In bubonic plague the process of diffusion is only shown by tumefaction in and around a gland at some distance from the primitive focus of inoculation. The process of diffusion is only shown by the successive tumefaction of lymphatic glands in parts more and more distant from the original focus of infection, and each swollen gland seems independent of its neighbour. If for instance the initial bubo is crural, in the course of two or three days and even less, inguinal buboes make their appearance, but they remain for many days perfectly distinct from each other, nor is any connecting inflammatory tract between these glands apparent.

THE BUBO THE ORIGINAL LESION.

In the great majority of cases the occurrence of a plague bubo is not preceded by any previous cutaneous injury that would serve to indicate the spot of penetration of the germs through the skin. Occasionally a trifling papule or phlyctene is found located as a rule on the antero-external aspect of the legs or on the dorsum of the foot. Still more rarely is a furuncle or ulceration seen, but whatever local lesion may be met with no intermediate alteration in the skin, lymphatics and veins, are to be observed. It is true that a lymphangitis has been described, but it occurs only when open sores have existed for some time, and when in the discharge pyogenic germs — staphylococci and streptococci — have developed. Plague lymphangitis is therefore always secondary, and when it appears it is quite late in the disease. Adenitis from tuberculosis, syphilis, &c., have distinctive characteristics which differentiate them at once from plague buboes, and with the exception of simple acute adenitis ending in abscess, and adenitis due to local venereal infection, there is not likely to be much confusion.

In connection with these two infections the nature of the bubo, the local pain, the temperature, the

symptoms of general infection, must be taken into consideration. In all other acute inflammations of lymphatic glands local swelling and pain precedes the fever, but in plague the fever, as a rule, precedes the local pain and the formation of the bubo. In plague we find violent headache, a temperature of 39° to 40° C., local lancinating pain becoming more and more acute, whilst as yet local glandular swelling is in abeyance. This prodromal period usually continues for twenty-four hours. The glandular swelling, when it appears, increases in size, until by the fourth or fifth day of the disease it attains the dimensions of a hen's egg; for a short time it remains stationary, being painful, hard and isolated from the surrounding tissues. There is no fluctuation, nor is the skin red or hot.

On the second and third day of the illness, whilst the bubo is increasing in size, symptoms of blood poisoning develop with stupor, delirium, &c. The tongue is coated, presenting livid red edges, the breath is fœtid, and the conjunctiva hyperæmic. Should a favourable issue result, delirium and tachycardiac symptoms in many cases continue after the temperature has been normal for nine days. The explanation of this phenomena is to be found in the fact that through the direct action of the leucocyte, the infective process is stopped owing to the destruction of plague bacilli; there however remains in the initial bubo a quantity of toxic products, which cannot be easily eliminated, and by finding their way in the blood continue to infect the system. In weak persons, the slow action of the toxin existing in the bubo may set up plague cachexia with fatal termination, if a rational cure is not adopted by timely incision of the bubo.

POSITIVE SIGNS AND SYMPTOMS OF PLAGUE.

The signs and symptoms of bubonic fever are of so destructive a character as to admit of no confusion between acute adenitis, even when the bubo is cervical. The characteristic features are as follows:—

(1) Fever with lancinating pain and some slight engorgement of a lymphatic gland in the groin, the axilla or the neck.

(2) Progressive and rapid tumefaction of the lymphatic gland or glands in these neighbourhoods; each gland forms a separate enlargement, and is hard and painful, well defined in outline, moveable on the deeper tissues, and under the skin, which has no share in the inflammatory process.

(3) Grave symptoms of general blood poisoning, out of proportion to the local lesion.

(4) In plague buboes no suppuration takes place during the period of infection.

Only after the crisis of the illness has passed, and when convalescence obtains, does softening of the glandular swelling take place, and spontaneous necrosis of the skin. As a general rule, however, the glandular enlargement disappears very gradually, and without suppuration. In very exceptional cases, owing to the infection of the swelling by germs other than plague bacilli, local symptoms may resemble simple adenitis, but in true plague the glandular swellings are much larger in bulk and in number, and the general symptoms are much more severe. Taking all these facts into consideration, a clinical diagnosis of bubonic

plague may be fairly positively arrived at, at any rate with sufficient certainty to justify steps being taken to deal with the case, and with suspects and outcasts, without waiting for further confirmation. In all cases, however, it is necessary to test the initial cases in any outbreak, bacteriologically and microscopically before pronouncing definitely upon the disease.

SEPTICÆMIC PLAGUE AND GASTRO-INTESTINAL CATARRH DUE TO PLAGUE.

As a rule cases of plague septicæmia and pneumonia appear only when cases of bubonic plague have already occurred, and the practitioner's attention is directed to the possibility of their ailments being due to plague infection chiefly by the gravity and severity of the signs and symptoms. Primary plague septicæmia is uncommonly rare and my experience leads me to affirm that it does not exist. Septicæmia in plague is always preceded by either gastro-intestinal catarrh or by a bubo, however small and imperceptible that bubo may be.

Septicæmic cases of plague are more apt to occur when the initial bubo is situated either on the neck or in the axilla; this is due no doubt to the more immediate continuity of these regions with the deep lymphatics in the thoracic mediastina, facilitating thereby a rapid diffusion of the bacilli or either organisms.

My experience teaches me that, even in the gravest cases of the septicæmic forms with a rapid course and before the appearance of the specific bacilli in the blood, a more or less evident tumefaction of some lymphatic gland accompanied by lancinating pain will be found. At autopsies of persons dead of septicæmic plague hypertrophied and hæmorrhagic axillary cervical glands infiltrated with plague bacilli are always found. These glandular enlargements cannot be admitted to be secondary to a general infection because they are commonly in limited numbers and in precise regions. It is therefore evident that from a pathogenic and clinical point of view, no distinction can be made between bubonic and septicæmic plague; the latter is only an acute and late stage of the former.

(To be continued.)

At a meeting of the Dermatological Society of Great Britain and Ireland, held on Thursday, April 24th, 1902, Mr. George Pernet showed some microscopical sections of Yaws (Paranghi of Ceylon). He was indebted to Sir William Kynsey for the original material. The sections were from a formed yaw. By Pappenheim's method the characteristic plasma-cells of granulomata in the corium were well brought out. There were also numerous leucocytes, which also invaded the rete of the epidermis. As to the latter, the basal layer showed pigmentation, as the yaw was obtained from natives of Ceylon. This pigmentation was best seen at the edges of the preparations. There was downgrowth of the epidermis, here and there cutting off portions of corium. There was also marked parakeratosis, the horny layers being here and there separated. The appearances differed in some respects from what is observed in syphilis and other granulomata.

TREATMENT OF LEPROSY BY INJECTIONS OF CHAULMOOGRA OIL.—In 1894 a leper was given 128 hypodermic injections of chaulmoogra oil during seven months; by the treatment the function of the sweat glands, which had been wholly lost, was restored. In 1896 the same man was treated by 106 injections of the same oil; the normal colour of the skin reappeared, and the fingers could be straightened. In 1897 injections to the number of 87 were administered; 50 injections were given in 1898, and 33 in 1899. During the whole period of treatment 90 ounces of oil were administered. At the end of the treatment, with the exception of a single nodule over the ulna, no trace of leprosy was left.—*Rev. de Therapeut.*

A CLINICAL NOTE CONCERNING TROPICAL HEAT-STROKE. By Dr. Salvatore Micela.—Observations on the clinical aspects of heat-stroke as it occurred in Italian soldiers in Africa. The symptoms observed were a sudden sense of oppression in the region of the heart, or of a constriction in the epigastrium; a very high temperature, a dry, hot skin, a weak rapid compressible pulse, a weak or absent apex beat, somnolence or coma, a pale face with semi-dilated pupils showing a slow reflex to light. In some soldiers there occurred bilious vomiting, trismus, convulsions, hallucinations, and delirium. The patients were all stripped and surrounded with ice, especially on the chest and the back along the vertebral column, and in addition received injections of caffeine. During a night march across a hot and sandy plain such cases were not observed, while in other marches under the burning sun, sunstroke occurred in a large number of soldiers. The hyperpyrexia came on insidiously, and seized the patient suddenly. The soldier, if not seen in time, strayed behind his comrades and fell on the ground, in some cases dead. In speaking of the pathological mechanism of this accident, the author sums up by saying that the excessive heat of the body paralyses the heart and the sensorium. Physiology teaches us that in mammals the heart beats cease when the body temperature reaches 44.5° C. It is possible, however, that the tropical region in which these heat strokes occurred was particularly favourable to such accidents on account of the great amount of decomposing vegetation capable of producing toxins that would be inhaled by the soldiers, or it is possible that the malarial parasite may have something to do with the intoxication of sunstroke. The author distinguishes heatstroke from sunstroke, and says that these conditions are frequently confused. Sunstroke occurs, even during repose, as the result of the direct action of the rays of the sun upon the head and neck. It consists in a congestion of the cerebral centres, and is accompanied by asphyxia and not by any increase in the temperature. On the other hand, heat-stroke occurs when there is added to the exposure to heat an excessive amount of muscular activity. It consists chiefly in a toxæmia which results from the disturbance of the compensating mechanism of heat regulation, and is accompanied by cardiac distress, a rise of temperature, and a lack of secretion of sweat.—*Gazzetta Degli Ospedali e Delle Cliniche*, March 16th, 1902.

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THE

Journal of Tropical Medicine

JULY 15, 1902.

HOW TO DEAL WITH THE OPIUM QUESTION IN CHINA—A SUGGESTION.

THE letter from Dr. James L. Maxwell in connection with the export of opium from India to China which is published in our issue of July 1st is well timed. The letter was a reply to statements contained in a leading article on the subject which appeared in this Journal on June 2nd.

It is a pity to see such high contending parties as the governments of Great Britain and of India on the one hand, and a section of the medical profession on the other, accusing each other of mis-statements, inaccuracies, and deliberate shuffling of responsibility. It is not so very long ago that one section of medical men stamped the work of the commission on opium, before which many medical men in India and China gave evidence, as "prejudiced garbage." We are afraid

no good can come of this line of argument. We are quite conversant with the evils of opium consumption in China, and whilst deploring the consequence, we are also ready and willing, as far as lies in our power, to assist in the campaign against the exportation, in quantity, of Indian opium to China and elsewhere. There is, however, a dignified method of conducting such a campaign, and one which from a rational standpoint will commend itself to every one.

The facts are these: Opium is being exported from India to China; the Chinese employ opium for the purpose of opium-smoking; the consumption of opium is detrimental to the physical and moral welfare of the Chinese; the Chinese wish to impose a duty on opium imported from India so as to lessen the evils arising from the consumption of the drug in China; the imposition of this duty is prevented by Foreign Governments. That is really the case as it stands, and if widespread support is to be enlisted, let the endeavour be to allow the Chinese to impose what tax on opium they please, seeing that such a step is necessary for the welfare of their country. Leave the guiltiness of the governments of Great Britain and India and the process of vituperation which has prevailed out of the question. The battle has not been won, nor is there any prospect of winning it, on the lines of recrimination. Were the question of the freedom of the Chinese to tax an imported article which is working destruction amongst them, brought forward by itself, there would be but few who would not assist to get the freedom granted.

In this way only permanent good might ensue, for as sure as India ceases its exportation other foreign nations will supply the Chinese markets. Little or no good will be done to China by proving that British ministers and authorities are guilty of breaking their promises. The whole question ought to resolve itself into what is to be done to save the Chinese from the deleterious influences of opium smoking, and the method which will attain that end is the one deserving of support. Will some of our readers frame a resolution embodying the proposition "that the Chinese be allowed to impose what tax they

please on imported opium." If this is done the well wishers of China—and they are many—will support the scheme heartily, and we will, if desired, circulate the resolution for signature amongst the widely scattered readers and subscribers of this Journal so as to obtain their signatures and those of their friends. A resolution of this kind backed by the signatures of responsible men and women would carry weight, and should be submitted not only to the Government of this country but to every Government in Europe and America.

J. C.

Translations.

A CONTRIBUTION TO OUR KNOWLEDGE OF THE PATHOLOGICAL ANATOMY OF PERNICIOUS SWAMP FEVERS (MORE ESPECIALLY AS REGARDS THE CHANGES IN THE PIA MATER).

By DR. A. PEWNITZKY,
Military Surgeon, St. Petersburg.
(Translated from the Russian.)

DR. A. PEWNITZKY has written a comprehensive monograph on the above subject, the result of his studies and observations in the bacteriological laboratory of the hospital for nervous and mental disorders attached to the Imperial Academy for Military Medicine, St. Petersburg.

The conclusions arrived at by the author, are as follows:—

(1) The pernicious attacks of swamp fever are particularly serious cases of malarial disease which are almost exclusively originated by the tropical tertian parasites.

(2) The severe course of the disease is caused by the remarkably luxuriant growth of this parasite in the organism, induced by conditions which are still unknown.

(3) The pernicious form of swamp fever runs a typical course, and, like the æstivo-autumnal fevers it commences in the middle of June and continues to October or November.

(4) This course of the disease depends on the circumstance that in malarial regions with hot summers the mean temperature of the day rises to from 25° C. to 27° C., this heat favouring the development of the tertian parasite in the stomach of *Anopheles*.

(5) Persons coming from fever-stricken districts between July and October and who are taken seriously ill, should be regarded as likely to have acquired pernicious swamp-fever, and an examination of the blood by an experienced medical man should immediately be undertaken.

(6) The study of the question of the clinical varieties of pernicious swamp fever, in conjunction with the increase of our knowledge as to the excitants of many infectious diseases, reveals that some of these varieties are of complicated origin as has been proved to be the case in the presence simultaneously of malaria and typhoid, malaria and cholera, malaria and croupous pneumonia.

(7) In the malignant forms of swamp fever the endothelium of the vascular walls are primarily affected by the effects of a toxin the nature of which is still unknown; the red corpuscles which harbour the sporozoa then find considerable resistance to their passage through the diseased capillaries, and severe circulatory disturbances are thus originated. These two conditions cause disorder of nutrition of the parenchymatous elements of all the important organs: such as the brain, the myocardium, the liver, spleen and kidneys.

(8) The changes in the cerebrum and cerebellum in the malignant forms of swamp fever are severe hyperæmia of the vessels of the pia mater, œdema, dilatation of the subarachnoid perivascular and pericellular spaces, engorgement of the capillaries (the endothelium of which is swollen) with a mass of disintegrated red blood corpuscles, which, in consequence of their increased circumference and diminished elasticity, traverse the capillaries slowly, and occasionally completely engorge them. This circulatory disturbance causes a number of punctiform hæmorrhages in the grey cerebral matter and at the junction of the grey with the white matter of the brain; it also causes various stages of degenerative and necrotic processes in the nerve-cells.

(9) In the pernicious forms of swamp fever phagocytosis is distinctly visible in the capillaries of the entire nervous system, and quite particularly in the spleen, bone, marrow and liver; the blood of the hepatic vein is, however, free from parasites.

(10) Besides the large mononuclear leucocytes, the cells of the adenoid tissue in the spleen, the lymphatic glands, and in the bone-marrow exhibit increased phagocytic activity, as does also the endothelium of the blood-vessels. This activity is not brought into play simultaneously, but develops energetically, sometimes in one direction, sometimes in another.

(11) The abundant development of the parasite in pernicious swamp fever is responsible for the fact that all the sporozoa are in the same stage of development simultaneously, and if it happens that the phase is the intra-corpuscular one in which quinine is ineffective, it is easily understood why the quinine treatment of such patients does not always save them. On this account a method of treatment must be adopted that is calculated to expel the toxin from the organism. For this purpose the subcutaneous injection of physiological solution of common salt may be employed, and quinine may be given in addition, as in this form it is quickly absorbed.

(12) As the cardiac muscle is seriously affected in the malignant forms of swamp fever, the use besides quinine of a sufficiency of cardiac stimulants such as caffeine is indicated in addition to the physiological solution of salt.

THE CLAYTON PROCESS OF DISINFECTING SHIPS.

THE Glasgow steamer "City of Perth," left Calcutta on May 4th, 1902, with a mixed cargo for Dunkirk, France. Shortly after leaving Malta, dead rats were discovered in a storeroom, and were handled by the steward and one of his assistants, both of whom contracted plague and succumbed to the disease.

The steamer was put in quarantine at Dunkirk and kept there over ten days, the French Sanitary Authorities at that port being quite unable to deal with the vessel. The owners were informed that the steamer would have to proceed to the Quarantine Station at St. Nazaire, and there discharge the cargo into lighters, and be subjected to eleven days quarantine afterwards. The owners were naturally very unwilling to incur this enormous expense and delay, and after considerable negotiation, permission was obtained from the authorities to bring the steamer to the Thames, to be disinfected, with her cargo on board, by the Clayton Company, who have a fumigating barge in London for this purpose. On the steamer arriving in the Thames, and after the cabins, &c., had been disinfected by the Port Sanitary Authority, "Clayton gas" was pumped into every part of the vessel where plague rats might be lurking. By this operation it will almost certainly be found that all rats on board, as well as the fleas, &c., with which they are infested, and which are the recognised conveyors of plague from rats to human beings, have been destroyed. The plague microbe itself succumbs to about three hours' exposure to the "Clayton gas," and every part of the vessel was subjected to the treatment for a considerably longer period, so that all danger of the disease spreading is averted. The cargo was subsequently discharged into lighters, and all dead rats discovered were cremated in the ship's furnaces. In a recent vessel disinfected by the Clayton process, some 1,500 dead rats were discovered after the operation.

Obituary.

MAXIMILIAN FRANK SIMON, C.M.G., M.D.

THE death of Dr. M. F. Simon removes from amongst us a distinguished officer of the Colonial Medical Service. His work in Singapore, where he spent the greater part of his professional career, was characterised by a thoroughness and conscientious devotion to duty, which no doubt hastened his end. For many years Dr. Simon held the important and responsible position of Principal Medical Officer of the Straits Settlement, during which period he initiated and developed many well-timed and radical improvements in the medical service of that important district. On his retirement from the service he had the honour of receiving a C.M.G., a well-earned distinction, but one which he was destined to enjoy for all too short a time.

It will be remembered, by those who were present at the meeting of the British Medical Association at Cheltenham, in 1901, that Dr. Simon was Secretary to the Section of Tropical Diseases, where he not only carried out the secretarial duties in an efficient manner, but contributed largely, from the wide store of his experience, to the scientific and professional success of the meeting.

For the approaching meeting of the Association in Manchester Dr. Simon was nominated as one of the vice-presidents, and it is needless to say that the Section of Tropical Diseases will be deprived of one of its most eminent supporters. As marking the thoroughness of Dr. Simon's professional work, it may be mentioned that he took out, in addition to his medical and surgical degrees and qualifications, the diploma of Dental Surgeon, feeling that, without a practical knowledge of dentistry, he could not adequately fulfil the duties of his position, in a colony where professional dentistry was at the time unknown. Of a gentle and retiring disposition his loss is keenly mourned by those who knew him intimately. Dr. Simon's writings on beri-beri, cholera and plague, stamp his professional knowledge and powers of investigation as having been of a high order; and these, together with his administrative abilities, obtained for him the honourable position which he occupied in the Colonial Medical Service.

Current Literature.

QUININE AND ITS SUBSTITUTES.

Substitutes for quinine are being assiduously sought for, with but doubtful success. In the *Centralbl. f. innere Med.*, 1901, the subject is discussed under the heading of "Quinine and its Esters," and several productions which are intended to obviate the undesirable effects of quinine are brought forward. An ester is technically pronounced to be "any compound ether which has an acid and an alcohol radical" (*Amer. Illustrat. Med. Dict.*). Euquinine = quinine-carbonic-ester is held to be a production which meets the want supplied. Other derivatives are acetylquinine, benzoylquinine, phosphorylquinine, saloquinine. The first-named is not of practical therapeutic use owing to its taste, the second and third are well nigh inert substance. The fourth on the list, saloquinine, not to be confounded with salicylate of quinine, acts as a mild quinine, and has some merits as a germicide, but its chief use is stated to be its analgesic effects in neuroses and neuralgias. A compound termed "rheumatin," a salicylate of saloquinine, is stated to be highly valuable in acute articular rheumatism.

CHOLERA.

CHOLERA IN THE PHILIPPINES.—Cholera has, for a considerable time, been reported prevalent in the Philippines. Although its ravages have been noticed chiefly amongst the natives the American soldiers have not wholly escaped.

The report received from General Chaffee by the War Department at Washington, on June 22nd, shows that the army in the Philippines is seriously affected by the cholera raging there. The report referred to states that twenty-two enlisted men died of cholera in the two weeks between April 23rd and May 6th.

Despatches sent out from Manila on June 21st stated that cholera was spreading in the islands. Thirty-five cases were reported in Manila on June 19th and twenty-eight deaths. The totals up to June 21st were 1,490 cases, and 1,197 deaths in Manila, and 6,959 cases and 5,098 deaths in the provinces.

This news is of a very grave nature, for if cholera of the type common in the far east gains a foothold among the American troops, the probability is that it will spread with great celerity and cause a large mortality.

Asiatic cholera is one of the most dreaded diseases of the East, and shares with the plague the undesirable notoriety of claiming more victims than any other malady of that region. However, the United States Military Medical Department of the Philippines may be relied upon to take every step known to sanitary science to stamp out and to curtail the spread of the disease.—*Med. Rec.*, June 28th, 1902.

The Fifty-third Annual Meeting of the American Medical Association was opened in Convention Hall, Saratoga Springs, N. Y., on Tuesday, June 10th, 1902, at which the following subjects were discussed.

Amœbic Dysentery IN MICHIGAN.—Dr. George Dock, of Ann Arbor, reported the case of a farmer who had not been out of Michigan for nine years and who developed chronic diarrhœa in the summer of 1901. The drinking water was not examined but was not obviously contaminated. Examination showed that the numerous small stools almost always contained mucus, blood, some leucocytes, and occasional small and superficial sloughs. In the mucus there were almost always Charcot's crystals and amœbæ, the latter usually in large numbers. The amœbæ measured from 20 to 30 microns in diameter; they contained red blood corpuscles almost always, and showed the characteristic motion. The patient's blood serum did not react with a culture of the *Shiga bacillus* furnished by Professor Flexner. No characteristic bacilli could be cultivated from the stools. Quinine enemata were almost entirely without effect on the process. Sublimed sulphur, in from 40 to 60 grain doses a day, had some effect on the number and character of the stools, and under its use the amœbæ became less numerous but did not entirely disappear. This was the first case of amœbic enteritis the writer had found in Michigan. In a case of ulcerated carcinoma of the rectum a smaller amœba was found without enclosed blood corpuscles. In a large number of cases treated with Carlsbad salts, amœbæ were never found, and the writer denied the truth of the assertion frequently made, that amœbæ were common parasites in healthy men. The writer discussed the relation of amœbæ to enteritis and urged the need of further work in American dysenteries.

Dr. McCrae, of Baltimore, said that several years ago he had seen one case occurring in a man who had never been out of the State of Maryland; this contradicted the assertion that all cases came from the south. In Maryland the disease was not infrequently found. A point of increasing interest was the fact that the disease was being more often noted in children, the majority of cases coming from the habit of many of these children of drinking the impure water from the street gutter.

Dr. James J. Walsh, of New York, had seen two such cases occurring in New York.

Dr. E. Libman, of New York, mentioned the frequent occurrence of liver abscesses at the Mt. Sinai Hospital, New York.

Dr. Dock, in closing, said that he did not include in his paper such cases occurring in soldiers who returned from Cuba or the Philippines.

TROPICAL FRAMBESIA AND TINEA IMBRICATA. By John T. Bowen, M.D., Boston.

Professor Koch¹ contributes some interesting facts about these tropical affections, gathered during his travels in New Guinea and the neighbouring groups of islands of the South Sea. He speaks of the lack of good pictorial representations of these affections in the literature, and is able to contribute several valuable photographs.

With regard to *frambesia*, it is said to be very extensively found throughout the tropics, appearing in many parts of Africa, in the East and West Indies, in the Indian Archipelago, and in China. It is somewhat doubtful, however, if the type is the same in all of these regions.

The *frambesia* seen in the South Seas is a contagious affection and can be inoculated from one person to another. One attack produces immunity. In places where it is endemic it is chiefly seen in children, as almost all people are attacked once. In certain places children are inoculated with it, in order that they may have it quickly and lightly, as was long ago the practice in the case of variola. Most of the children seen by Koch were between one and twelve years of age. The lesions are ulcers approximately circular in form, often grouped or confluent. They always project above the surface of the skin, and appear as luxuriant granulations lying upon the skin. The smallest lesions looked not unlike a variola pustule, with a marked umbilication in the centre. The larger lesions were always denuded of epidermis, exuded a serum and pus, and were covered with moist crusts, which exposed, upon removal, the exuberant granulating mass. When situated near the anus or genitals they resemble condylomata very closely, and are sometimes mistaken for the latter. In the same child the most varied stages of the individual nodules may be met with, including small nodules still covered with epidermis, and all stages of ulcerated nodules. The lesions are especially frequent

¹ Arch. f. Derm. u. Syph., January, 1902.—*Boston Med. and Surg. Journ.*, June 12, 1902.

about the mouth and genitals. The paper is illustrated by several good photographs. The nodules do not all appear at once, but from time to time, until the susceptibility of the patient for the disease has been overcome. The duration is from some months to a year. Infants or very young children frequently die of the disease. It is not known that Europeans are ever affected. Various micro-organisms have been described as causing the disease, but none have been substantiated.

Tinea imbricata also seems to find its chief habitat in the South Sea Islands, and to have spread out from there to China and the Straits Settlement. This is caused by a parasite similar to the *Tricophyton tonsurans*, which grows in the rete, and like ringworm develops in the form of circles, but does not tend to heal in the centre, so that annular appearances are not produced. The epidermis exfoliates in small scales, which remain adherent by their peripheral part, and present somewhat the appearance of the tiles of a roof, whence the name *imbricata*. There is a considerable amount of pruritus. In the photographs large surfaces of the body are covered with the circular scaling lesions, which produce a most extraordinary appearance. One remarkable picture is that of a child showing the earliest manifestations, where the sternal region is the seat of an enormous circular lesion. The affection is very common in adults, and sometimes almost all the inhabitants of a village may be affected. In adults, as a rule, almost the whole body is affected, so that Koch believes that the affection makes its appearance at a very early age, spreads gradually over the whole body, and, as far as he can determine, never heals spontaneously. It appears to have no essential effect on the general health.

PLAGUE.

PREVALENCE OF THE DISEASE.

INDIA.—During the weeks ending June 7th, 14th and 21st, the deaths from plague in India numbered 2,473, 1,316 and 1,294 respectively.

EGYPT.—During the weeks ending June 15th, 22nd, 29th, and July 6th, the fresh cases of plague in Egypt were returned as 4, 5, 8 and 11; and the deaths from the disease as 1, 2, 7 and 7 respectively.

CAPE OF GOOD HOPE.—The number of fresh cases of plague for the weeks ending June 7th, 14th and 21st were reported to be 1, 1, and 0; and the deaths from the disease as 0, 2 and 0 respectively.

HONG KONG.—During the weeks ending June 28th, July 5th and 12th, the fresh cases of plague numbered 44, 38 and 28; and the deaths from the disease, 43, 31 and 27 respectively.

CONSTANTINOPLE.—On June 30th, 4 cases of plague were reported from Constantinople; 1 death was announced.

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Archives Russes de Pathologie, de Médec. Clinique et de Bacteriologie.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.

British Medical Journal.
Brooklyn Medical Journal.
Caducée.
Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Esercito.
Hong Kong Telegraph.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
Journal of the American Medical Association.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal
Medical Brief.
Medical Missionary Journal.
Medical Record.
Medical Review.
Merck's Archives.
New York Medical Journal.
New York Post-Graduate.
Pacific Medical Journal.
Polyclinic.
Public Health.
Revista de Medicina Tropical.
Revista Medica de S. Paulo.
Sei-i-Kwai Medical Journal.
The Hospital.
The Northumberland and Durham Medical Journal.
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- 4.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.
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In order to meet the constant enquiry for addresses of respectable firms catering for the various requirements so difficult to obtain abroad, we give a list of names and addresses which we trust will be found useful to our numerous correspondents and subscribers.

Original Communications.

STUDIES IN PLAGUE.

By Professor C. TERNI.

Of the Bacteriological Institute of Messina, Italy.

(Continued from page 228.)

TEMPERATURE IN PLAGUE. DISTURBANCES OF THE RESPIRATORY AND CIRCULATORY SYSTEMS.

EXCEPT when a furuncle or other local trouble occurs at the seat of inoculation no increase of temperature is observed in plague, until the lymphatic glands, the lungs, or the intestinal tract give evidence of being affected. An initial febrile period, ushered in by rigors, obtains, in which the temperature rises quickly to 39° or 40° C., and continues within these limits, with short remissions, for from three to six days. This period, during which the primitive bubo is formed, is succeeded by a fall by crisis, or in cases in which the bubo is reabsorbed or suppurates the fall is gradual. During suppuration the common pyogenic germs may by their presence cause an elevation of temperature, which ceases when the bubo is opened. What I call the spontaneous suppuration of the plague bubo in mild cases is not attended by pyrexia or the presence of pyogenic bacteria in the pus. When, however, as in grave cases, the infection extends to lymphatic glands other than the initial, the temperature continues to rise to 39° or 40° C., and this, the second period of the illness, ends in death or in protracted recovery. The second period might correctly be termed the septicæmic, for it marks the passage of the infection from the primary bubo to the circulatory system, and the temperature shows oscillations in direct proportion to the occurrence of new plague foci. In the gastro-intestinal form of the disease the same fluctuations occur as fresh mesenteric glands become involved in the bubonic lesions. In the acute gastro-intestinal form of plague, after

a short period of high fever, collapse occurs, and the temperature remains low until death occurs. In pneumonic plague and in the septicæmic form the temperature, after a short remission, keeps high until death occurs. In all acute cases, however, the temperature constantly shows an initial period of fever, followed by a slight fall, to be followed again by marked remissions. It is this period of subsidence of fever which proves delusive, and is wont to be attributed by the inexperienced as evidence of recovery, resulting from treatment by medicines, by plague serum, &c.

It is remarkable that secondary febrile accessions due to plague are never accompanied by rigors, nor are they attended by perspiration; these are characteristic symptoms in febrile conditions due to the presence of pyogenic germs, and their absence in plague amount to a diagnostic feature.

The Disturbances in the Circulatory System commence early. The pulse increases in frequency to 120 to 130 beats per minute, independently of an increase in temperature. During the first few days of the illness it keeps strong, full and elastic; when the disease becomes general the frequency is still greater, but the sphygmographic curve diminishes in range until the waves of accession of all are reduced to well-nigh a straight line. A dirotic pulse is common in the early periods of the pneumonic, the septicæmic and the gastro-intestinal forms of the disease.

The contrast between the pulse-rate and the thermic curve when collapse occurs is marked, for we find that in very low temperatures the pulse rises to 160 to 180 or over.

Even during convalescence and for a month after the cessation of fever the pulse frequency may continue at 120, probably until all absorption from the focal lesion has ceased.

Removal of the bubonic glands causes an immediate fall in the pulse-rate; this phenomenon indicates extirpation of the primary gland as a surgical step of

imperative necessity, being a much more reliable therapeutic agent than doubtful antitoxin remedies.

The gravest cardiac lesions are those of a paralytic nature, which apparently are due to degeneration of the plague bacilli. It is found in these paralytic states that the bacteria are vacuolised or have degenerated into a disc, or ring-like shape, and exhibit few of the characteristic microscopic features of the live bacillus of plague. It would appear as though the protoplasm of the bacillus had escaped by dialysis through the bacterial wall and set up toxic changes, resulting in paralysis of the cardiac muscle.

Experiments by Lustig and Galeotti and by myself show that the poison extracted from plague bacteria and with juices filtered from buboes and organs in plague cases directly cause circulatory disturbances, and that even the delirium and stupor met with are due to circulatory rather than to nervous disturbance. The lesion does not, however, lie in the heart muscle or in the nervous supply of the heart, but in the effect the poison has on the vaso-motor system. It is a case of vaso-motor paralysis, which chiefly manifests itself in the capillary innervations; the heart paralysis being secondary.

MICROSCOPIC EXAMINATION.

The following method of staining and preparing specimens for microscopic examination of the plague bacillus gives good results:—

(1) Spread a thin stratum of the juice obtained from a bubo, from the excreta, the vomit, the organs or tissues of dead bodies, or of the blood on a slide; should the specimen be coagulated dilute with a drop or two of distilled water.

(2) Dry over a flame with moderate heat.

(3) Fix the specimen by exposing it for from three to ten minutes to Roux's fluid (equal parts of absolute alcohol and ether); dry again by moderate heat.

(4) Colour with the following fluid: distilled water 100 parts, crystallised phenic acid 2 parts, saturated alcoholic solution of gentian and violet or fuchsin 10 parts. Of this solution add 20 to 30 drops to 30 cm. of distilled water and with it stain the preparation for a few seconds.

(5) Wash well with distilled water, dry and mount the specimen permanently.

With the plague bacilli in certain stages of the disease may be found pyogenic micrococci, streptococci, the diplococcus of Frankel and, more rarely, the coli-bacillus, &c., but to an experienced bacteriologist these accessory bacteria can hardly cause confusion.

The diplococcus is more likely to cause hesitation in arriving at a positive conclusion than any other form of bacterium. In the *gastro-intestinal* catarrh due to plague, the bacilli will be found gathered for the most part into zooglae, adherent to the epithelium or flakes of mucus; on the other hand they may appear as a chain or almost as a pure culture.

Plague bacilli do not appear in the blood as a rule until the disease is well advanced and even then they are usually few in number. When they are met with in the blood the plague bacilli are usually found to be well vacuolised, and occur sometimes isolated, sometimes in pairs, or, again, in chains of three or four elements; they present, moreover, an evident capsule,

so much so that they may be mistaken for diplococci. As both the diplococci and the plague bacilli present great variability in form and size it is always well to employ Grassi's method as a means of differential diagnosis.

In *plague pneumonia* the bacilli are usually present in extraordinary quantity and are commonly associated with diplococci. The plague bacilli may occur in the pneumonic form singly, or gathered into zooglae, and are never encapsuled.

Bacteriological Examination.—In glycerine agar culture medium (3 per cent.) at a temperature of from 30° to 35° C., the plague bacillus produces colonies, visible to the naked eye, within twenty-four hours. At first the colonies resemble those produced by streptococci, or diplococci, but microscopic examination will at once clear up the diagnosis. As a further test of the diagnosis it is requisite that the effect upon animals be tried. To accomplish this: sow a portion of the juice gathered from a bubo in tubes of glycerine bouillon (3 per cent.) and inject it by the syringe into the peritoneal cavity of a guinea-pig. In twelve hours the peritoneal fluid of the guinea-pig may show a prolific growth of plague bacilli. This test holds good even when the bacilli in the blood, or juices used, are very scanty.

In coming to a conclusion as to the diagnosis of plague by microscopic and bacteriological examination, the following points are to be noted and observed:—

(1) The plague bacillus has well-defined specific morphological characters.

(2) There is no acute adenitis produced by germs which can be mistaken for bubonic plague.

(3) Acute adenitis, when the clinical characters of plague are present, require to be investigated bacteriologically.

(4) The microscopic and bacteriological diagnosis of plague are founded on positive scientific data.

(5) When it has been established by bacteriological examination that plague is present in a locality, the clinical symptoms are alone sufficient to establish a diagnosis.

(6) The characteristic signs and symptoms of plague are:—

(a) Fever, ushered in by rigors with lancinating pains in a lymphatic gland or glands, and subsequent swelling of the painful gland or glands.

(b) Increased frequency of the pulse independently of a rise in temperature.

(c) The bubonic swelling is tense, movable under the skin and on the deeper tissues, non-fluctuating and painful on palpation; by the 3rd or 5th the bubo attains its maximum size.

(d) Symptoms of a general toxic state do not correspond with the changes and condition of the primary glandular lesion. Of all signs and symptoms to be noted in plague the nature of the pus from the bubo and the character of the pulse are of most consequence. The more sanious the pus the more virulent is the disease, and as long as there is a focus of suppuration, and consequently a liability to general blood poisoning by the plague bacillus, so long will the small and frequent pulse obtain; this marked feature in regard to the pulse continues even when the temperature is

almost, or wholly, normal. In mild cases in which rapid suppuration occurs, the specific bacillus of plague disappears from the pus and even from the walls of the tubo after two or three days.

THE THEORY OF THE CAUSATION OF BERI-BERI BY A TOXIN CONVEYED BY RICE, CONSIDERED IN THE LIGHT OF LOCAL EXPERIENCE OF THE DISEASE.

By E. A. O. TRAVERS, M.R.C.S., L.R.C.P.

State Surgeon, Selangor.

(Published by permission of the Colonial Office.)

THE THEORY ORIGINATED IN JAPAN.

THE theory to the effect that the consumption of certain kinds of rice plays an important part in the causation of beri-beri, was, I believe, first promulgated by the Japanese, in connection with an extensive outbreak of the disease in the Japanese Navy.

Recently, special attention has been drawn to this theory by Dr. Braddon, State Surgeon of Negri Sembilan, who, in a paper read before the Conference of Medical Officers of the Federated Malay States, held in 1900, and again in a Memorandum on the subject published by Government in 1901, very strongly advocates this theory, which he formulates as follows:—

THE THEORY AS FORMULATED BY DR. BRADDON.

"The original cause is assumed to be a mould or microbe which grows upon padi (and possibly other grain) in the places in which beri-beri is endemic. When the padi has been reaped and stored, the mould, or its spores, remained undestroyed (like ergot in rye, or the cause of pellagra in maize, or of lathyrism in vetch), and may possibly continue to grow and multiply. When such padi is husked or milled, the spores fall into the *bras*, where, under favouring circumstances—chief among which are probably warmth and moisture—they grow, and produce a ferment or toxin. The consumption of the *bras* or rice so poisoned is that which is conceived to be the cause of beri-beri."

IMMUNITY OF TAMILS FROM BERI-BERI THE MAIN ARGUMENT IN SUPPORT OF THEORY.

Dr. Braddon's main argument in support of this theory is that Tamils, or natives of southern India, who form about 83 per mille of the population of Selangor, very rarely suffer from beri-beri.

This immunity Dr. Braddon refers to as follows:—

"To what cause can this protection be ascribed?"

"In the answer to this question lies the key to beri-beri.

"In reviewing the habits of life of the different races which might be conceived to affect the genesis or incidence of a disease such as beri-beri, one and only one factor appears as a constant difference between Tamils and others, which seems in any way likely to be able to account for his protection from the disorder: it is that the staple food of the Tamils throughout these States, as also of the 70 or 80 millions of these fellows in their native country, differs from that consumed by every other nationality.

"That food is rice, but it is rice which is, in fact,

prepared before use in a manner not customary with any of the other races, and by a process which forms, indeed, a more or less efficient method of sterilisation.

"The rice eaten by Tamils, grown in whatever district, is prepared from padi which is always either steamed or scalded in the husk, and thereafter dried before milling. Rice prepared in this manner is known as Bengal rice.

"By such a process any poisonous fungus growing in or upon the husk of the grain would naturally be destroyed, so that there would remain during the milling no contaminatory germ or body which could fall into the rice so as to be able afterwards to grow there and to give poisonous properties to it. Such a factor, and no other that has hitherto been suggested, will, if my view of the causation of beri-beri as an intoxication be correct, account for the protection of the Tamils from a disease which decimates all around them."

With the object of illustrating the marked immunity from beri-beri enjoyed by Tamils in these States, Dr. Braddon gives a number of figures, the more important of which are as follows—(I have taken the figures for Selangor only):—

| "YEAR 1900. | |
|--|--------|
| "Estimated Tamil population | 14,244 |
| "Proportion of Tamils per 1,000 of estimated total population | 83 |
| "Proportion of Tamils per 1,000 admissions to State Hospitals for Beri-beri | 9" |

TAMILS ALSO ENJOY AN IMMUNITY FROM LEPROSY.

These figures conclusively prove that Tamils enjoy an extraordinary immunity from beri-beri; but they also enjoy an almost equally remarkable immunity from leprosy, as may be seen by the following figures:—

| | |
|--|------|
| Number of Lepers admitted to Asylum during last five years | 512 |
| Number of Tamil Lepers admitted during the same period | 9 |
| Proportion of Tamils per 1,000 of estimated population | 83 |
| Proportion of Tamils per 1,000 admissions to Asylum for leprosy | 17.3 |

As the bacillus of leprosy is now well known, I do not think that the advocates of the rice theory of beri-beri would be prepared to say that leprosy is caused by a germ or toxin generated in a form of rice which is not eaten by Tamils. If, however, they are not prepared to account for the immunity of Tamils from leprosy in this way, then little importance can be attached to the same theory with regard to beri-beri.

OTHER DIFFERENCES BETWEEN HABITS OF TAMILS AND OTHER NATIONALITIES.

In considering the difference between the habits of life of the Tamils and other nationalities which can account for the immunity from beri-beri of the former, it is difficult to see why only the food supply should have been mentioned. The fact that Tamils regularly anoint themselves from head to foot with gingelly oil, in taking what they call the oil-bath, is quite as striking a difference between the habits of the Tamils and those of other nationalities, and is as likely to protect them from beri-beri as a slight difference in the method of preparing their rice.

Then, again, the Chinese, who are the chief sufferers

from beri-beri, are in the habit of watering their vegetables with liquid human manure. This unsavoury custom is peculiar to the Chinese, and might be advocated as a cause of beri-beri, and as a reason why the Tamils do not suffer from the disease.

It will thus be seen that Dr. Braddon attaches too much importance to the fact that Tamils enjoy a marked immunity from beri-beri, inasmuch as they enjoy practically the same immunity from leprosy.

It is a well-known fact that certain diseases are peculiar to certain races, and that, on the contrary, in many places a certain race will not suffer to any extent from a disease which may be very common among other nationalities in the same country.

OUTBREAK OF BERI-BERI IN THE PUDOH GAOL.

In the month of August, 1895, the prisoners incarcerated in the large local prison known as the Pudoh Gaol, were severely attacked with beri-beri, which has since caused a large number of deaths, and has continued, with occasional slight intermissions, up to the present time.

An opportunity has been thus afforded for making many observations and experiments with regard to the disease, some of which are of great importance in connection with what may be called, shortly, the Rice Theory of the Causation of Beri-beri.

DIET OF PRISONERS.

The diet of the Chinese prisoners, as well as of the patients in the various large hospitals in the district, consists mainly of so-called Rangoon rice, with fresh vegetables and either fresh pork or salt fish.

The food is supplied by a Chinese contractor who purchases his rice from merchants in Penang. The rice arrives in bags, and at no one time has more than 150 bags, or about a three weeks' supply, been imported. The Penang merchants procure the rice from many districts, but mainly from Rangoon. The rice for the prisoners is sent to the Pudoh Gaol at intervals of about three to four days.

It is cooked by steam, which is forced through it at a high pressure by means of a small vertical engine.

The Pudoh Gaol was occupied in January, 1895, previous to which all prisoners were confined in what may be called the Old Gaol, Kuala Lumpur, about a mile and a half distant. While in the Old Gaol none of the prisoners contracted beri-beri, although they were fed on the same kind of rice, procured from the same source as that given them when in the Pudoh Gaol.

This is well shown by the following figures:—

| Year | Rice Supply | Cases of Beri-beri Admitted to Infirmary | Deaths from Beri-beri | Remarks |
|------|---|--|-----------------------|--|
| 1892 | Old Gaol Rangoon rice from same source | 6 | .. | These cases were admitted with the disease, and did not contract it in the gaol. |
| 1893 | | 2 | 1 | These cases were not contracted in gaol. |
| 1894 | | 8 | .. | These cases were not contracted in gaol. |
| 1895 | | 158 | 34 | Contracted in gaol. |
| 1896 | Pudoh Gaol | 478 | 42 | " " " |
| 1897 | | 275 | 54 | " " " |

APPARENT CAUSE OF OUTBREAK: CHANGE OF LOCALITY AND NOT RICE SUPPLY.

It is evident, therefore, that in this case the outbreak of beri-beri occurred immediately after a change in the location of the prisoners, the supply of rice remaining the same.

The case mortality from beri-beri in the Pudoh Gaol Infirmary being extremely high, and in view of the apparent influence of locality on the disease, all patients suffering from beri-beri were transferred to the Old Gaol on October 1st, 1895, in the hope that the change of residence might prove beneficial. The result of this arrangement is shown as follows:—

| Period | No. of Beri-beri Cases Admitted | Per-centage of Deaths | Remarks |
|---------------------|---------------------------------|-----------------------|--|
| 1895 { September .. | 32 | 31.7 | Beri-beri cases treated in Pudoh Gaol. |
| October .. | 35 | 15.57 | |
| November .. | 23 | 6.15 | |
| December .. | 25 | 4.25 | |
| | | | Beri-beri cases treated in Old Gaol. |

The food for the beri-beri cases treated in the Old Gaol was not only exactly similar in every respect to that used in the Pudoh Gaol, but it was actually cooked in the Pudoh Gaol with the food for the other prisoners, and was conveyed in a handcart to the Old Gaol.

Encouraged by the evidently beneficial results of change of locality on the sick prisoners, arrangements were made to experiment still further in this direction. On October 21st, 1895, sixty prisoners showing no signs of beri-beri, and in apparently good health, were transferred from the Pudoh Gaol to the Old Gaol; from this date to July, 1896, a large gang of prisoners were confined in the Old Gaol.

The monthly average number of prisoners in each gaol during this period, with the number of fresh cases of beri-beri occurring among them, is shown by the following figures:—

FIGURES SHOWING MONTHLY AVERAGE NUMBER OF PRISONERS IN EACH GAOL.

| Month | Average No. of Prisoners in Pudoh Gaol | No. of Beri-beri Cases occurring in Pudoh Gaol | Average No. of Prisoners in Old Gaol | No. of Beri-beri Cases occurring in Old Gaol |
|-------------------|--|--|--------------------------------------|--|
| 1895 { October .. | 297 | 88 | 72 | Nil. |
| November .. | 337 | 21 | 119 | " |
| December .. | 271 | 28 | 128 | " |
| January .. | 280 | 29 | 133 | " |
| February .. | 286 | 47 | 130 | " |
| 1896 { March .. | 271 | 43 | 119 | " |
| April .. | 275 | 42 | 97 | " |
| May .. | 299 | 36 | 106 | " |
| June .. | 287 | 39 | 84 | " |

FOOD SUPPLY THE SAME IN BOTH GAOLS.

From October 1st to December 14th, 1895, all food supplied to the healthy prisoners in the Old Gaol, as well as to the beri-beri patients transferred from the Pudoh Gaol, was, as has been stated above, cooked in

the Pudoh Gaol, with the food for the other prisoners, and carried to the Old Gaol twice daily, the diet being exactly the same at both gaols, and the rice being taken out of the same bag and cooked in the same steamer. After December 14th, 1895, the rations for the prisoners in the Old Gaol were cooked in that institution, raw rations, with the exception of rice, being sent daily from the Pudoh Gaol. The rice was sent direct from the contractor to the Old Gaol, where it was kept in bags and used as required. It may, I think, be fairly claimed that the opportunity for experiment with regard to the influence of food supply on the etiology of beri-beri presented by the outbreak at the Pudoh Gaol has been an exceptional one, and that full advantage has been taken of it.

NO CONNECTION BETWEEN RICE SUPPLY AND OUTBREAK OF BERI-BERI.

The results of these experiments and observations seem to indicate very clearly that in, at any rate, this instance there was no connection of any kind between the outbreak of beri-beri and the food supply.

According to the theory formulated by Dr. Braddon, it is assumed that a mould or microbe forms on the padi when growing in a place where beri-beri is endemic; that when the padi is husked or milled the spores fall into the rice, and there, under favouring circumstances, they grow and produce a ferment or toxin, which is conceived to be the cause of beri-beri.

It will be seen that the whole chain of events is assumed, and that no detail of the theory is based on actual observation or practical test. If, however, the mould, the spores, and the toxin are taken as actually existent, it is extremely difficult to apply the theory to our experiences in Selangor.

There are in our chief town, called Kuala Lumpur, three other large institutions, under the care of the Medical Department—the District Hospital containing about 450 beds, the leper asylum with 130 beds, and a hospital for incurables containing 60 beds. These institutions and the Pudoh Gaol are within three miles of each other, and the conditions of temperature, moisture of air, &c., are practically the same.

The rice supplied for the inmates of these institutions is of the same kind, and is obtained from the same contractor, as that used in the Pudoh Gaol. The contractor assures me that the rice as received from Penang is distributed to the different hospitals, and that in no case is any of the rice kept in Kuala Lumpur for more than three weeks.

Throughout the six years during which beri-beri has been endemic at the Pudoh Gaol there has been no outbreak of beri-beri at any of the institutions mentioned.

If, therefore, the rice infected in Rangoon, or wherever it grew, was the cause of beri-beri among the prisoners in the Pudoh Gaol, why did it not cause a similar outbreak in the Leper Asylum, District Hospital, and Hospital for Incurables?

If, however, it be assumed that the rice was either not infected, or at any rate the toxin was not produced in it, when it reached the Pudoh Gaol, but that this occurred during the period (at no time exceeding four days) during which the rice was kept in bags at

the gaol, then the immunity of the other institutions might be accounted for. The fact, however, that the prisoners transferred to the Old Gaol enjoyed complete immunity from beri-beri, although fed on rice from the same source and actually cooked in the same steamer as the prisoners in the Pudoh Gaol, who were suffering severely from the disease, effectually disposes of this supposition.

The outbreak of beri-beri at the Pudoh Gaol has presented many other extremely interesting features which it is not necessary for me to mention here, but which will be given in detail in a history of beri-beri in the Gaol now being written by Dr. Hamilton Wright and myself.

I have in this memorandum only mentioned those circumstances which bear especially on the rice theory of the causation of beri-beri. I have done this in the hope that our experiences in Selangor may be of use to those now occupied in making investigations into the etiology of this interesting disease.

The case of a Chetty, who developed beri-beri at Malacca during the year 1901, is of considerable interest in connection with the "rice theory" and the causation of beri-beri. This man was under my treatment in Kuala Lumpur, and the case was carefully observed by Dr. Hamilton Wright and myself. The Chetty was a high-caste Tamil of considerable wealth, and during the three years he had resided in the Straits Settlements he had never eaten any but the very best Bengal rice (*Puloonga arisi*) imported from India by the Chetties themselves.

The case was a very typical one of paralytic beri-beri, and is of interest as an instance in which a Tamil contracted beri-beri although he had never eaten any but Bengal rice, especially prepared in the way mentioned by Dr. Braddon.

SANITARY REPORT OF HONG KONG FOR THE YEAR 1901.—The Medical Officer of Health for the colony of Hong Kong, Dr. Francis W. Clark, has favoured us with a copy of the Annual Sanitary Report. The report is drawn with care and with a scientific accuracy which renders reports of the kind valuable additions to medical literature.

Malarial Fevers.—The death rate amongst the Chinese from malaria amounted to 1·9 per 1,000.

Beri-beri.—Of the Chinese, 377 persons died of beri-beri; the deaths were distributed during the year as follows: January, 26; February, 34; March, 14; April, 22; May, 26; June, 16; July, 23; August, 40; September, 47; October, 44; November, 51; December, 38. Dr. Clark is inclined to attribute beri-beri to infected food, and most probably to damaged rice or other grain affected by fungoid growth.

Plague.—During the year 1,562 persons died of plague; yet in spite of the persistence of plague in the colony, the death rate shows a considerable reduction during the past ten years as compared with the previous, the former standing at 27·81 per 1,000, the latter at 23·42 per 1,000.

BIRTH.

On July 23rd, at 26, Upper Phillimore Gardens, Kensington, London, W., the wife of Dr. Percy Athelstan Nightingale, of Harrogate, of a son.

Journal of Tropical Medicine.

PRIZE ESSAYS ON SUBJECTS CONNECTED WITH TROPICAL DISEASES.

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LADY MACGREGOR,

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THIS PRIZE WAS NOT AWARDED.

Judges—Surgeon-General Roe Hooper, C.S.I., President Medical Board, India Office; Colonel Kenneth MacLeod, LL.D., Professor of Clinical and Military Medicine, Netley; Patrick Manson, C.M.G., F.R.S., LL.D., Medical Adviser, Colonial Office and Crown Agents of Colonies.

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THE

Journal of Tropical Medicine

AUGUST 1, 1902.

British Medical Association.

SECTION OF TROPICAL DISEASES.

INTRODUCTORY REMARKS MADE AT THE ANNUAL MEETING OF THE BRITISH MEDICAL ASSOCIATION, HELD IN MANCHESTER, JULY—AUGUST, 1902.

By Sir WILLIAM R. KYNSEY, C.M.G. F.R.C.P.I.

President of the Section.

It affords me great pleasure to preside over this Section, and the honour conferred upon me by the British Medical Association in appointing me your President is one I highly appreciate. I accepted the position, I may assure you, as a compliment to the Colonial Medical Service, in which I spent many years of my life.

It is the usual custom for the President to open the scientific business of this Section with some introductory remarks on matters of interest. When I remember the high professional standing and the discoveries of those who have preceded me in office, I feel constrained to ask your kind indulgence for any defects that may be apparent in the observations I address to you. I have read with interest and instruction the addresses which have been delivered from this chair, and I find it impossible to choose

a topic in connection with tropical medicine which has not been already ably touched upon by my predecessors.

By the Memorandum of the Association for the guidance of officers of Sections, the Presidents are wisely requested not to deliver a formal address in opening their Sections, so as not to interfere with sectional work, and that any introductory remarks should not occupy more than fifteen minutes. I need hardly state that under such instructions it would be impossible for me, even if I possessed the necessary knowledge, to review the advances of tropical medicine during the past year, or to dwell on our ignorance of the causation of many of the ordinary diseases met with in tropical practice.

In the words of Lord Salisbury on a memorable occasion, "We live in a small bright oasis of knowledge surrounded on all sides by a vast, unexplored region of impenetrable mystery. From age to age the strenuous labour of successive generations wins a small strip from the desert and pushes forwards the boundaries of knowledge"; or, as that great philosopher, Professor Huxley, well puts the same idea, "The known is finite, the unknown infinite: intellectually we stand on an islet in the midst of an illimitable ocean of inexplicability; our business in every generation is to reclaim a little more land, to add something to the extent and solidity of our possessions."

Marvellously minute observation is perhaps the most notable feature of scientific research in the last quarter of the nineteenth and the opening years of the twentieth centuries, combined with a severe spirit of criticism, and that now, as has always been the case, science, like religion, metes out its rewards only to those who diligently seek it. In medicine one of the most surprising discoveries has been the relationship which has been proved to exist between insects and grave disease—the direct outcome of the modern spirit of research.

I have no intention of occupying your time with an account of the successive malarial discoveries made by observers of different nations which can now be found in the text-books, and are known even to the man in the street, nor with the unworthy questions too often raised by discussions about priority. But it cannot be too often repeated that in the whole story of medical science there has been recorded nothing more wonderful than the prophylactic measures which have followed on the discovery of the malarial organism by the illustrious Frenchman, Laveran, and by the investigations into its life-history by Italian, German, and American observers, and by our own countrymen, Manson and Ross, according to the experimental method advocated by Bacon and Harvey. If these investigators have no other reward, they will have the highest satisfaction men of science can enjoy—that of extending our knowledge of disease and of doing good to humanity. I consider the discovery of the malaria parasite—and that man is its temporary and the mosquito its definite host and transmitter, that it completes asexual life and prepares its sexual forms in human blood, while it completes the sexual cycle of life, that by which the life of the parasite external to man is assured in a particular species of mosquito, and that man becomes infected only through

the bite of the *Anopheles*—one of the most epoch-making events of the age in which we live. It may be truly said that scientific research has gone hand in hand with practical and preventive medicine. The physician can give quinine with a full knowledge of how his remedy acts; and the sanitarian can try, and in many cases succeed in preventing the occurrence of malarial diseases by methods devised on scientific principles. The treatment and the modern prophylaxis of malaria exemplifying the Greek idea of Socrates and Plato that "right knowledge involves right action."

Another most gratifying result of the study of the causation of malaria, and directly traceable to it, is the increased interest taken in the investigation of all tropical diseases by the establishment of schools of instruction, and the formation of scientific expeditions and travelling scholarships. In the schools of tropical medicine of London and Liverpool, and from the lectures on diseases of the tropics in many of our colleges, the medical man intending to practise abroad can acquire an acquaintance with the diseases he will be called upon to treat, special advantages long enjoyed by the army, Indian, and naval services in the great schools of Netley and Haslar. I have the most grateful recollection of the instruction I received at Netley, where diseases from all parts of the world could be seen and studied. No one who had the privilege of their acquaintance can forget the gifted and amiable Parkes, the father of hygiene; that accomplished gentleman speaker and writer, Maclean, whose lectures may still be read with profit; and Aitken, the first pathologist of his day, whose work is a storehouse of facts relating to tropical medicine. It is pleasant for an old Netley man to feel that the instruction in that great school has not deteriorated, and that it continues to send forth highly-trained officers, ever ready to fight disease in any part of the world. If the rumour is true that the army school is to be soon moved to London, I am sure you will all join me in wishing it a brilliant future.

In the history of the past year there is no great discovery to be recorded; it may be looked back upon as one of steady progress in elucidating some of the many problems of tropical medicine, the greatest and most beneficent being in the scientific prevention of disease. In the case of yellow fever the mosquito—a *Culex*, not *Anopheles*—has been tried and found guilty of being the sole agent in the spread of that disease. In Havana, yellow fever was endemic for a century and a half; during the past year it has been freed from the scourge by killing the mosquitoes in the neighbourhood of each focus of disease as discovered, and by carefully disinfecting every house that had lodged a yellow fever patient in order to destroy the mosquitoes that had bitten a sick person. This great sanitary triumph must ever redound to the honour of American medicine.

No excuse is necessary in this section for dwelling a little on the important subject of the prophylaxis of malaria. An epidemic of any kind is among the most costly in life and money that can befall a town or district. An epidemic of malaria differs from other outbreaks of disease in this important fact, that its effects are not limited to the deaths it causes, but

that it often leads to years of suffering, poverty, and depopulation.

I have high authority for stating that, taking one year with another, malaria destroys twice as many people in India as cholera, small-pox, and all other epidemic diseases put together. It is a matter of history that the same cause almost annihilated the soldiers of the Walcheren expedition, and the army under Wellington in Spain was so assailed by malaria that, according to Ferguson, the enemy and all Europe believed the British forces were exterminated. Malaria stopped the great Panama scheme of de Lesseps. I have known districts in Ceylon almost depopulated. A most disastrous outbreak occurred some years ago at Galle—a town previously malaria free—attended with great loss of life, in consequence of the construction of a railway, the embankments of which were made by excavating pits at the sides that soon were filled with rain-water. In those days the origin of the fever was, of course, attributed to disturbance of soil. We now know the pits provided breeding places for the *Anopheles*, and the Sinhalese and Tamil labourers from malaria districts supplied the organisms.

Although we were ignorant of the true conditions which caused the outbreak, we advised the filling up of the pits on the disturbance of soil theory with satisfactory results, proving that much can be done in the prevention of disease before full knowledge of causation is reached.

I will only occupy a few minutes of your time while I describe two experiments—one made by the Colonial Office on Dr. Manson's advice, and the other by the Japanese Government, which clearly show it is both possible and practicable to prevent malarial infection.

In the experiment conducted by Drs. Sambon and Low in the Roman Campagna and in London, in order to prove the truth or error of the mosquito malarial theory, and also how far protection against malaria might be consistent with the ordinary avocations of life, two experiments were necessary—one that healthy persons in London should be bitten by infected mosquitoes from a malarious region, and the second that men should live in an undoubtedly malarious place during the fever season, being protected only from the bites of mosquitoes. Those bitten in London contracted ague. Drs. Sambon and Low and two others lived in a mosquito-proof hut in a most intensely malarious place in Italy, where all the inhabitants suffered from malaria. The four inhabitants of the hut remained perfectly healthy all the time, and, I believe, are so still.

The experiments by the Japanese Government were carried out on the Island of Formosa, and they furnish a most conclusive demonstration of the relations between mosquitoes and malaria. A battalion of soldiers who were completely protected from mosquitoes for one hundred and sixty-one days during the malaria season escaped the disease entirely; whereas, there were 259 cases of malaria in another battalion in the same place and during the same length of time not protected from mosquitoes.

I am sure you will agree with the conclusions of Drs. Sambon and Low that their experiments prove

STAFF OF HASLAR HOSPITAL AND JUNIOR OFFICERS ATTENDING THE COURSE OF INSTRUCTION DURING THE TERM ENDING JUNE 20th, 1902.

[illegible]

that mosquitoes only are capable of transmitting malarial fever; that protection from their bite implies absolute immunity; and that protection can be easily obtained. If further proof is considered necessary for large bodies of men, it is furnished by the Japanese experiment.

By a study of the life-history and surroundings of the *Anopheles*, and by varied and long-continued experiments, Ross has determined the best means of preventing malarial infection. He advised the extermination of the *Anopheles*, and although this is difficult it is not so difficult as at first sight it appears. These mosquitoes breed in small pools of a certain kind easily recognised and easily dealt with, always close to human habitations, as the females must pass frequently between the pools where they lay their eggs and the houses where they obtain their food. If the *Anopheles* are found in a house the breeding puddles are close by. The greatest practical points derived from Ross's work are two:—

(1) That the life of the *Anopheles* is in direct and intimate relation with the annual epidemics of malarial fever.

(2) That it is only necessary to drain the *Anopheles* puddles and not the whole of a malarious district.

I believe the discovery of the malarial parasite, and the investigations into the life-history of the mosquito which conveys it to man, have placed us in the position to suggest measures which, if carried out, would have the effect in an unhealthy district of largely reducing the amount of malarial fever and eventually of exterminating it.

(1) Malarial fevers should be included and notified among infectious diseases, so that precautions could be taken to prevent their spread.

(2) Persons, especially children, suffering from malaria, primary infections or recurrences, should be isolated and treated with quinine to prevent, as far as possible, the infection of mosquitoes.

(3) Persons infected with malaria should be prevented coming to a healthy place to infect mosquitoes.

(4) The puddles in which the *Anopheles* breed should be drained and filled up, or treated with kerosine to destroy the larvæ.

(5) Doors and windows of houses, gaols and hospitals in malarious districts should be screened with wire gauze, and beds should be provided with netting to prevent mosquitoes entering and biting the occupants.

I am painfully aware how far this address falls short of what might have been accomplished by a more skilful handwriting upon the all-important subject of malaria prevention, but my remarks will not be thrown away if they induce those not acquainted with recent work to take an interest in and to appreciate the wonderful results in tropical medicine which have followed on the discovery of the malarial parasite.

Let knowledge grow from more to more,
But more of reverence in us dwell;
That mind and soul according well,
May make one music as before:
But Vaster.

July 30th, 1902.

Sir WILLIAM R. KYNSEY, President, in the Chair.

DISCUSSION ON BERI-BERI.

I.—PATRICK MANSON, C.M.G., F.R.S., introduced the subject of beri-beri for discussion, by stating that we should be in agreement as to what the word beri-beri indicates. It has been applied to ankylostomiasis, to epidemic dropsy, to sleeping sickness, and even at the present day it is possible a variety of ailments are erroneously grouped with beri-beri.

As from under the term malaria we are gradually weeding out diseases which have long been falsely associated, so in time beri-beri will be freed from an associated group of ailments with which it has nothing to do. Clinically, beri-beri is a multiple peripheral neuritis. From the etiological standpoint, in the tropics as elsewhere, there are many kinds of peripheral neuritis, which, although different in their causation, have much in common clinically. Undoubtedly in the tropics we find cases of peripheral neuritis arising from alcohol, from ptomaines of different kinds, from minerals, such as tin and arsenic, and from organic poisons developed in the body in the course of specific infections. Indeed, beri-beri has lately been regarded as mere arsenical poisoning; but without entering into the argument, there are many clinical and epidemiological arguments to show that arsenical neuritis and peripheral neuritis are distinctly different conditions. In regard to malarial neuritis, although rare, it is possible there is such a thing, but the great majority of these affections are really beri-beri. Beri-beri may co-exist with malarial parasites in the blood just as it may co-exist with tuberculosis. Dr. Manson quoted examples to prove both. He also drew attention to the peculiar loss of memory that appertains to severe malarial infection, and contrasted this with the clear intellectual state of beri-beri patients.

DISTINGUISHING FEATURES OF BERI-BERI.

From other pathological groups with neuritis as their leading clinical phenomenon beri-beri is distinguished by (1) our ignorance of the cause; (2) its occurrence as an endemic and epidemic disease; (3) its proneness to produce cardiac disability and dropsy; (4) the non-implication of the cranial nerves with the exception of the pneumogastric; (5) the non-implication of the intellectual and emotional centres; (6) the rarity and complete absence of trophic skin lesions; (7) the high rate of mortality under certain conditions.

In considering the etiology of a disease it is convenient to divide the subject into (1) the immediate cause—germ, toxin, inadequate and improper food, traumatism, or whatever it may be; (2) the means by which, or the medium through which, the cause is applied; (3) the circumstances personal to the individual which influence his receptivity and susceptibility; (4) the physical conditions external to the patient, favourable or the reverse, to the application and operation of the cause.

Assuming that we are dealing with only one form of neuritis in what we call beri-beri, I hold that this neuritis is produced by (1) a toxin; (2) the toxin

is the product of a germ operating in some culture medium located outside the human body; (3) the toxin enters the body neither in food nor in water, but through the skin or by inhalation.

The germs which from time to time have been discovered in beri-beri patients have been discredited, and never more completely than by the recent work of Dr. Stanley, of Shanghai.

That the toxin of beri-beri is produced by a living germ is proved by the fact that the disease can be introduced into virgin countries and there spread. The hypothetical cause is capable of being transported and of multiplying. Spontaneous multiplication is a property peculiar to living things; therefore the originating agent of the toxin of beri-beri is a living thing—a germ.

The question of infection by rice has held the field of argument for many years. But although disputed by many, it has never been completely refuted until the present moment. Recent papers by Dr. Bolton, of Diégo Garcia, and by Dr. Travers,¹ of Kuala Lumpur, prove this up to the hilt. Dr. Travers' paper marks an epoch in our knowledge of beri-beri, for though his results are entirely of a negative character, they effectually sweep away a mass of crude conjecture, and narrow down very much the field for future investigation.

The inference to be drawn from this conclusive experiment is that beri-beri has no direct, if any, connection with food, most certainly not with rice. He further, however, specifies that "all food" supplied to the beri-beri and non-beri-beri patients was bought, cooked, and distributed from the same sources, and as we know that water has nothing to do with the etiology of beri-beri, it is safe to conclude that neither food nor drinking water play a part in the production of beri-beri.

II.—E. R. ROST, Captain I.M.S., contributed a paper on "The Cause of Beri-beri."

During an outbreak of beri-beri in a gaol in India, Captain Rost observed that pigeons living in large numbers under the roofs of the gaol buildings were affected by an epidemic disease which caused paralysis of the wings and death. At this time Captain Rost thought he had traced the disease to a micrococcus which he found in the Jewari—the staple food of the gaol. After preventing the pigeons visiting their home under the roof the epidemic died out. A favourable opportunity for investigation arising again in Rangoon, Captain Rost found in rice-water liquor and in mouldy rice an angular diplo-bacillus between the starch granules, and found that this was extremely resistant to high temperature. He found this organism also in the blood and cerebrospinal fluid of a large number of beri-beri cases, and cultivated it in broth, rice-broth and ascitic fluid.

Captain Rost subsequently experimented with the diplo-bacillus he describes, when he found fowls infected with the growth became paralysed and died; and the bacillus was found in their blood and tissues. He also suspects rice as the cause of the disease in man, and attests evidence which he describes "in itself is so remarkably in favour of the rice origin

as to leave little doubt as to the causation of the disease."

III.—L. W. SAMBON, M.D., failed to see how Dr. Travers' experiment swept aside the amount of evidence accumulated on rice as a possible factor in the etiology of beri-beri. He did not, of course, think that rice was pathogenic because deficient in mineral and nitrogenous matters, but that rice might be related in some places to beri-beri, as maize is to pellagra, or, in other words, that rice might become the vehicle of the disease by special contamination.

IV.—RONALD ROSS, C.B., F.R.S., said he had obtained further analyses of the hair from beri-beri patients, and that of 30 cases examined 10 had exhibited traces of arsenic. It was peculiar, also, that it was only in the most recent cases of the illness that the arsenic was found; in only one old case (five months' standing) was a trace found. Major Ross in no way affirms that beri-beri and arsenic stand to each other as cause and effect, but recent records have suggested the possibility, and the evidence cannot be lightly laid aside.

V.—C. W. DANIELS, M.B.(Camb.), stated that tropical peripheral neuritis included other diseases than beri-beri, especially a post-dysenteric peripheral neuritis. It is owing to the inclusion of so many ailments under the description of beri-beri that so many widely different conclusions were arrived at.

VI.—JAMES CANTLIE, M.B., remarked that he had seen beri-beri spread in a ward to surgical cases with ulcers, whilst the medical cases escaped. He hoped to find investigations in beri-beri carried out on the same lines as yellow fever had been investigated by the U.S. Army Commission in Cuba.

VII.—W. T. PROUT, M.B., Sierra Leone, said that beri-beri was not endemic in Sierra Leone, but he had seen a ship arrive on the coast from Panama with 250 out of 500 negroes affected by beri-beri. The negroes were landed and properly cared for. Only nine of them died, and the disease did not spread beyond the confines of the beri-beri camp.

VIII.—P. A. NIGHTINGALE, M.D., stated that beri-beri was not an endemic in Bangkok, and that outbreaks of the disease were very rare. He had, however, 18 months ago, seen a sharp epidemic of beri-beri at Bangkok.

IX.—Sir WILLIAM KYNSEY said that although beri-beri was considered to prevail at one time in Ceylon, he had never seen a case in residents or natives of the island; all the cases he saw were importations. He would like to have more definite information as to the popular tale of the consumption of arsenic by Styrian peasants.

Other papers read were:—

(1) *The Climate and Diseases of Bangkok*, by P. A. Nightingale, M.D., Harrogate.

(2) *Enteric Fever in Sierra Leone*, by Major F. Smith, R.A.M.C.

(3) *Dysentery*, by Andrew Duncan, M.D., F.R.C.S., F.R.C.P.

(4) *The Prevention and Treatment of Dysentery in Institutions in the Tropics*, by W. T. Buchanan, Major I.M.S.

¹ Dr. Travers' paper will be found at p. 231 of this issue of the Journal.

(5) *Tropical and Amœbic Abscess of the Liver, and its Relationship to Amœbic Dysentery*, by Leonard Rogers, Major I.M.S.

(6) *Sanitary Work in West Africa*, by M. Logan Taylor, M.B., Ch.B.

(7) *Bilharzia hæmatobia in Cyprus*, by G. A. Williamson, M.D.

(8) *Yellow Fever*, by James Cantlie, M.B.

(9) *The Differential Diagnosis of Yellow Fever and Malignant Malaria*, by G. C. Low, M.B.

(10) *The Agglutinating Properties in the Blood in Cases of Mediterranean Fever, with Special Regard to Prognosis; Remarks on other Blood Changes and Reactions during the Course of the Disease*, by P. W. Bassett-Smith, Staff-Surgeon R.N.

(11) *The Geographical Distribution of Malta Fever and the Value of Splenic Enlargement as a Test of Malarial Incidence*, by James A. Hislop, L.R.C.P., L.R.C.S., Assam.

(12) *Kala-azar, as an Analogous Disease to Malta Fever*, by C. A. Bentley, M.B., C.M., Assam.

(13) *Heat Apoplexy*, by Edward Henderson, M.D.

(14) *The Prophylaxis of Sunstroke*, by Andrew Duncan, M.D., F.R.C.P., F.R.C.S.

(15) *Contribution to the Study of the Bacillus pestis*, by Professor Galli-Valerio, Lausanne.

(16) *Remarks concerning the Nomenclature, Etiology, and Prophylaxis of the Intermittent Fevers*, by L. W. Sambon, M.D.

(17) *Malarial Fever in Cyprus*, by G. A. Williamson, M.D.

(18) *Filariasis in Sierra Leone*, by W. T. Prout, M.B.

(19) *Malta Fever in the Canaries*, by Brian Melland, M.D.

(20) *The Leucocytes in Malaria*, by Charles Melland, M.D., M.R.C.P.

(21) *A Trypanosome in Man*, by J. E. Dutton, M.B.

THE LIVERPOOL SCHOOL OF TROPICAL MEDICINE.

SIR ALFRED JONES, K.C.M.G., Chairman of the School, entertained at dinner at the Adelphi Hotel, Liverpool, on August 1st, 1902, His Grace the Duke of Northumberland, K.G., and the members of the Tropical Diseases Section of the British Medical Association. Amongst those present were: The Bishop of Liverpool; Sir Wm. MacGregor, K.C.M.G.; Sir George Denton, K.C.M.G.; C. McArthur, M.P.; Professor Clifford Allbutt, F.R.S.; Sir William Banks, F.R.C.S.; Hon. James Boyle, U.S.A. Consul; Professor John Chiene, C.B., F.R.S.; Professor Carter, F.R.C.P.; Professor Boyce, F.R.S.; Professor Stoeltzner; Mr. Rushton Parker, F.R.C.S.; and Dr. Grunbaum. The Members of the Section of Tropical Diseases present were: Sir William Kynsey, C.M.G.; Dr. Patrick Manson, C.M.G., F.R.S.; Major Ronald Ross, C.B., F.R.S.; Professor Sandwith, F.R.C.P., of Cairo; Professor Madden, F.R.C.S., of Cairo; Dr. W. T. Prout, of Sierra Leone; Dr. J. E. Dutton; Dr. C. W. Daniels, M.B.; and Mr. James Cantlie, F.R.C.S. The toast list was as follows: "The King," proposed

by the Chairman; "The Duke of Northumberland," proposed by the Chairman; "Tropical Medicine," proposed by the Lord Bishop of Liverpool, responded to by Sir William Kynsey, C.M.G.; Dr. Patrick Manson, C.M.G.; and Professor Clifford Allbutt, F.R.S.; "Our Tropical Colonies," proposed by the Chairman, responded to by His Excellency Sir William MacGregor, K.C.M.G., Governor of Lagos; His Excellency Sir G. C. Denton, K.C.M.G., Governor of the Gambia; and John Holt, Esq.; "The Chairman," proposed by Mr. C. McArthur, M.P.

Over one hundred guests sat down to dinner, and a most enjoyable evening was spent. The toast of "The Chairman" was ably given by Mr. McArthur, and most cordially received by the company.

Translation.

ON LOMADERA, A SPECIES OF EXCEEDINGLY WIDESPREAD TEXAS FEVER IN VENEZUELA.

By DR. HANS ZIEMANN, Naval Staff Surgeon.

(Translated from the German by P. Falcke.)

In the following article I report a few results of investigations on a cattle disease in Venezuela during my stay in that country whilst serving with H.M.S. "Moltke." My stay lasted from November 28th to December 15th, 1901, during which time I visited the more important places on the coast, and went by rail from La Guayra through the mountains to Caracas, then to La Victoria, and from Porto Cabello again I went as far as Valencia.

The cattle disease, the cause of which in these places was quite unknown and which was called "lomadera," or "ringadera" plague, is, it may be taken for granted, either "Texas fever" or a disease analogous to it. In 1897 I discovered a new centre of this disease in Northern Italy, and by means of my special method of staining the bacterium of the disease¹ I even at that time felt fully convinced that this disease, which is so fatal to cattle, must have a far larger region of distribution.²

On the present voyage, therefore, all my endeavours were fixed on gaining more material respecting this blood disease which in many particulars so nearly resembles malaria.

Incidentally, during the investigations, an extraordinary dissemination of distomum hepaticum was found to be present in the adult oxen on the islands off Cape Verdi; on the other hand, ticks—ixodidæ—which in actual Texas fever transmit the disease were conspicuous by their absence. However, during the course of a walk from La Guayra to Macuto, I came across a herd, the oxen of which were all infested by ticks.

¹ Ziemann, *Neue Untersuchungen über die Malaria und den Malariaerregern nahestehende Blutparasiten. Deutsche medizinische Wochenschrift*, 1898.

² In regard to my discoveries in Germany compare H. Ziemann, *Ueber das endemische Vorkommen der seuchenhaften Hämoglobinurie der Rinder in Deutschland. Deutsche medizinische Wochenschrift*, 1901, No. 21.

In Germany I only found ixodes reduvius on the cattle in the infected districts, but these ticks appeared to be identical with ixodes australis. It is evident that this nomenclature requires correction.

TEXAS FEVER PREVALENT IN VENEZUELA.

The investigations conducted in La Guayra, in the abattoir of Caracas, on the pastures near Valencia, and in the Hinterland of Porto Cabello, and even on board ship on an infected calf taken with us, led to the following astonishing result. That seemingly a large percentage of the native Venezuelan cattle suffer from "Texas fever" and that nearly all the imported cattle succumb to this fatal disease. This observation should be of scientific and practical interest. The classical investigations of the Americans, Theobald Smith and Kilborne, have revealed the fact that an area of distribution of Texas fever exists in the south of the United States, where even at the present day it does damage to the amount of £4,000,000 per annum; moreover, the investigations of Lignières have confirmed a second centre of distribution in the Argentine. By my discoveries those two centres of disease separated by large tracts of country are brought into closer local connection. The everlasting revolutions in these fertile countries have rendered scientific enquiry a matter of difficulty. A wealth of material awaits the investigator provided with modern appliances; for yellow fever, small-pox, malaria in its severest forms, blackwater fever, leprosy and the most interesting skin diseases are found; and in parts also, beri-beri, anchylostomum and filarial disease prevail, varying in frequency according to the locality. Thus, I was surprised to hear that blackwater fever is relatively frequent on the Orinoco. A few observations on this subject will be found elsewhere. In regard to the diseases of animals the countries mentioned are as yet *terra incognita*.

THE DISTRICTS INFECTED.

Venezuela was formerly not a country from which cattle was exported. The export of coffee and cocoa, and sugar in a less degree, were the sources of its prosperity. Since the culture of coffee has decreased through the considerable fall in its price, and impoverishment has further increased through the political conditions that prevail, more attention has been given to the breeding of cattle as a means of revenue. Porto Cabello and Guanta Barcelona are the export harbours for cattle; St. Jago de Cuba, Habana in Cuba, and Trinidad are the principal ports of destination of the cattle. The breeding of the cattle takes place inland on the immense high pasture lands, where the so-called guinea and gamelote grasses provide the animals with abundant food. In the west and south these pastures are bordered by enormous primeval forests watered by the tributaries of the Orinoco. In order to reach the sea-shore the herds must cross the fairly steep mountains to reach La Victoria and Valencia. I also saw fairly large pastures near Valencia and Lake Valencia. The forests on the mountains near the coast have all been burnt down, and the mountains now are but scantily wooded. The partial scarcity of water in the country is certainly attributable to this circumstance; as an instance I heard that in Cumana there is sometimes

no rainfall for three years. The actual dry season in the pasture land lasts from November to May. During February, March and April the cattle are not driven to the coast; the reason for this, as explained to me, was that during these months the cattle could not find enough food *en route* during their journey. June, July and August are the principal months for export, and in a less degree December, January and February. The railway is but little used for bringing the beasts to the coasts, as they always arrive in bad condition after the railway journey. It is customary to drive the cattle to the above-mentioned pastures near Valencia, which are mostly the property of the large exporters.

The minimum length of this journey is computed as being equivalent to a fourteen hours ride on horse back.

The cattle remain on these pastures for three or four weeks, sometimes for two or three months, to be fattened, and then are driven to the coast in two or three days, where they are left a day or so and then conveyed to Cuba by steamer. In Cuba the purchasers send them to grass for another two or three months before being killed. The cattle from the interior mostly reach the coast in a more or less deplorable condition, dependent largely upon the barbarity of the drivers and the scarcity of food on the way. I myself saw freshly arrived oxen in Caracas which were scarcely able to crawl owing to sore boofs. The animals that have already acquired the germs of disease often die during this fatiguing journey, so that a sort of natural selection has taken place amongst the cattle that reach the coast.

SIGNS AND SYMPTOMS OF LOMADERA.

It is particularly during the dry season that oxen—and it is said horses also—are attacked on the high pasture lands by a disease that mostly has a fatal termination. It is manifested first of all by languor, the sorry appearance of the animals, diarrhoea and disinclination for food. The symptoms may prevail a few days, whereupon hæmaturia sets in and the conjunctivæ become yellow. It was not possible for me to ascertain if a rise of temperature occurred, though it may be concluded that such was the case; it is improbable, also, that the temperature of a sick beast was ever taken in Venezuela. Often during the illness the milk and fæces were tinged with blood. The animals almost always perish from exhaustion. The duration of the illness fluctuates between a few hours and a few days; it is only rarely that the disease drags on for a fortnight.

POST-MORTEM APPEARANCE.

The principal changes observed in the cadavers as noted by me in the slaughter-yard in Caracas in animals that were seemingly healthy but in reality diseased, were as follows: The muscles are very pale and anæmic, the entire subcutaneous cellular and adipose tissues are tinged yellow, the kidneys present a condition of parenchymatous inflammation, the liver and spleen are enlarged, the gall bladder distended with bile, the bile ducts in the liver dilated and also full of bile. In one case also small punctiform extravasations of blood were observable in the intestine. In streak preparations, particularly from

the heart, kidneys and spleen, the blood parasite—the pyrosoma bigeminum to be mentioned below—was observable in great profusion. The shortness of the time at my disposal, the remarkable want of interest displayed by these southern people in the well-being of their cattle, as well as the fact that the cattle observed by me had passed through the hands of several drovers, all rendered the task I had in hand difficult; and it was impossible to ascertain whether the cattle could be attacked twice or thrice by the disease, and whether steers and oxen, or calves or cows are most liable. One fact appeared certainly to be known to all the drovers, namely, that the best and heaviest cows were most liable to be most severely attacked. I ascertained that in this country the cows only exceptionally yield 12 to 15 litres of milk per diem; the average yield is 1 to 2 or 3 litres.

ETIOLOGY.

The cause of lomadera, according to the local popular opinion, is that the heat and the direct rays of the sun combined with scarcity of food and water originated the disease. When an animal died it was at once buried. The pasture was then burned down and the cattle driven to another pasture.

Remedies seemingly were not applied. *It is noteworthy that the ticks are not brought into etiological connection with lomadera. Only so much was known, that a beast beset with many ticks became "sad." I succeeded, however, in confirming the fact, first, that the most extensive appearance of lomadera was contemporaneous with the enormous number of ticks that appeared in the dry season, and that the ticks, even in the coast regions, at least as regards La Guayra and Porto Cabello, are present in the underwood in great numbers. At every step through the undergrowth the female ticks may be found clinging to one's arms and legs. On the pastures of a Mr. Ermen, in Valencia, I saw young calves that had been born there covered with ticks. Indeed, nearly all the cattle in Venezuela were, according to my observations covered with ticks, and almost always to a much greater extent than the cattle with hæmaturia in Germany. In a drove of 800 at Valencia not one animal seemed free. All details, including the experiments with ticks taken off seemingly healthy and sick cattle will be reported on in a book I intend publishing on hæmaturia of cattle in Germany. As to this disease, which is considerably less destructive than lomadera, it may be mentioned that there are essential differences between the two diseases.*

THE PARASITES OF TEXAS FEVER AND LOMADERA COMPARED.

The exciting factor in lomadera, as found in the red blood corpuscles, are minute light bodies possessing unusually lively local movement with a diameter of from $\frac{1}{4}$ to 1 μ . When grown larger they become roundish formations with amoeboid movement; the larger, however, they grow the less motile do they become; they also become paler. The largest size of these roundish parasites observed by me averaged 2.5 to 3.0 μ in diameter. *The motility of the small parasites within the red blood corpuscles was maintained for an uncommonly long time—for about five or six hours in cover-glass preparations, and as long as eight days*

in red blood corpuscles in the contents of the stomach of ticks taken off a sick beast. Very frequently from two to six of the smallest parasites were present in one red blood corpuscle, sometimes also two larger round ones. It was not uncommon to observe one larger parasite to which one or two smaller ones were attached as if just segmenting. Often, also, annular forms were observed which microscopically were hardly distinguishable from the well-known annular forms of the tropical malaria parasites.

In one calf, that was permanently under observation, pear-shaped parasites also appeared, mostly two in each blood corpuscle in such a position that the two pointed extremities touched. The two together occupied about two-fifths of the capacity of the infected red blood corpuscle; they were therefore mostly larger than the corresponding forms in Germany. These pyriform bodies in Venezuela were also immobile. I cannot now enter into the question of the multiplication of the parasites. The details of the structure of this interesting parasite, as ascertained by the method of staining described by me elsewhere, entirely correspond with the description first given by me¹ of the parasites of Texas fever, and which was fully confirmed by other authors. The parasites consist of a small heap of chromatin, representing the nucleus surrounded by a light area and the protoplasm. When anæmia has set in in consequence of the disintegration of the numerous infected red blood corpuscles, or the toxic effects of the parasites, it is possible in lomadera also to observe basophile granules in numerous non-infected red blood corpuscles. As, however, Theobald Smith has demonstrated, the granules thus stained with basic colouring matter have nothing to do with the parasites.

It is interesting to note that of four cattle taken for examination haphazard in La Guayra, and which had been driven to the coast from the interior a week previously, three exhibited a considerable infection of the blood with small round parasites. The animals which were not thinner than the average, did not exhibit the slightest sign of ill-health. One was an exceedingly lively steer. All were very much infested by ticks. Of two seemingly healthy cows in Valencia infested by ticks, one likewise exhibited infection with small round parasites, as did also a calf seven days old, born there and very strong; so also did a calf born in Valencia, twenty days old. The latter, certainly, looked very miserable after having lost its mother, but was otherwise healthy; as the infection was very considerable it was taken on board for further experiment. On the following day it was taken with typical hæmaturia, probably as an indirect consequence of its railway journey and change of food (feeding by bottle), and after nine days it died, and an autopsy made.

From the above observations it appears to me that the practical conclusion come to is that first of all lomadera occurs not only on the high inland pastures but also in the districts nearer the coast, and that seemingly healthy cattle of this district may be the

¹ H. Ziemann—Ueber Malaria u. andere Blutparasiten, nebst Anhang eine wirksame Methode der Chromatin und Blutfärbung, Jena. G. Fischer.

bearers and transmitters of this disease. According to the assertion of the various drovers, lomadera is supposed not to occur at all at the localities I observed, to wit Valencia; at the season of my visit, however, the disease did prevail there, but it exhibited no visible symptoms. It may at once be gathered what importance this circumstance has for export or rather for the export harbours. According to my investigations in Germany, *pyrosoma bigeminum* is not capable of transmission from the mother (intra-uterine) to the calves, but my observations on the seven-days-old calf prove that infection in Venezuela can already take place within seven days. It is my firm conviction that the remarkable paucity of the milk of cows in Venezuela is due only to latent infection with lomadera. It is to be hoped that in more peaceful times a wise law, similar to the one obtaining in the United States, will place a check on this plague. It is even feasible at present by destruction of the ticks to make it possible to import better strains of cattle for the improvement of the native cattle, an experiment in this direction having been formerly made and relinquished on account of its costliness. I may here mention that I succeeded in Germany in procuring an exceedingly light infection in a series of sucking calves by means of inoculation of virulent blood, which afforded a high degree of protection against the subsequent natural infection.

In conclusion I will add a few words on lomadera of horses in Venezuela. Unfortunately, I was unable personally to observe a case. According, however, to descriptions of the disease, its course is still more rapid and more frequently fatal than in horned cattle. Clinically it appeared to correspond fairly well with the so-called "Kreuzrhehe" of horses in Germany. It may therefore be mentioned in this connection that I examined a horse in Oldenburg that had a slight attack of "Kreuzrhehe" accompanied by hæmaturia, and in examining the blood I discovered small parasites with lively movements, which exhibited no difference from the adolescent forms of the parasites of the hæmaturia of cattle. Unfortunately, I was unable to demonstrate my opinion that the "Kreuzrhehe" of horses is an infectious disease by inoculation of the blood of the sick horse into a healthy one.

Addendum—On St. Thomas, a Danish island of the West Indies, I also found that Texas fever was present, though not considerable; so that the regions of infection of the United States and Argentine receive another connecting link. According to accounts received, a disease similar to the Texas fever of horned cattle seems to prevail amongst the sheep on St. Thomas; this, perhaps, corresponds with the *carceag* of sheep described by Babes as occurring in Roumania.

In April, 1902, I succeeded in experimentally demonstrating that larvae which I had myself bred from the ova of the ixodes (*australis*), brought by me to Europe, originated "lomadera" in a healthy German calf from an immune district within seven days. The larvae grew into the adult state on the calf. I was unable to demonstrate this experiment with *ixodus reduvius*.

The tick in question has proved to be *Rhipicephalus annulatus* (Say), *Boophilus bovis* (Riley).—(*Deutsche Medizinische Wochenschrift*, 1902, Nos. 20 and 21.)

QUESTIONS CIRCULATED BY DR. A. VALASSOPOULO CONCERNING THE "TYPHUS BILIEUX" OF EGYPT.

SIR,—Having been deputed by the first Egyptian Congress to make a report on "*typhus bilieux*," a disease that is endemic in Alexandria, I venture to submit the following list of questions to you, and shall be obliged if you will send me the answers thereto.

I remain, &c., &c.,

DR. A. VALASSOPOULO,

Physician-in-Chief to the Greek
Alexandria Hospital.

QUESTIONS.

1. Have you observed any cases of "*typhus bilieux*" in the town you practise in?
2. Were they sporadic cases, or circumscribed outbreaks?
3. At which season of the year did they take place?
4. What were the predominating symptoms?
 - (a) On which day of disease did jaundice appear?
 - (b) Was the albumen in the urine abundant, and did this phenomenon appear early?
 - (c) Did the course of the fever and the nerve symptoms remind you of a "*typhique*" illness?
 - (d) Were the stools discoloured?
 - (e) Did your patients exhibit marked myalgia during the first day, and which group of muscles were most involved?
 - (f) Did your patients exhibit cardiac symptoms, and at what period of the disease?
 - (g) Have you made a special study of the urinary symptoms in this disease?
 - (h) Have you observed an urinary crisis at the close? Have you observed relapses?
5. What was the mortality? What was the immediate cause of death (syncope or uræmia)? At what period of the disease did death supervene?
6. Have you noted any particulars as to the etiology, the pathogeny, the diagnosis, and the treatment of "*typhus bilieux*"? Have you observed cases of contagion in the family of patients, or facts that would lead you to suspect transmissibility of the disease, direct or indirect? What is your opinion on this question? Have you examined your patient's blood for Obermaier's spirochæte?
7. Have you observed cases of "*typhus hepaticus benignus*" or relapsing "*ictère fébrile*"? The disease studied by Albert Mathieu, in Paris, and by A. Weil, in Berlin, which is called "Weil's disease in Europe"?
8. Do you believe there is identity between Weil's disease, "*l'ictère infectieux*," benign or severe, and the Egyptian "*typhus bilieux*," as described by Griesinger?
9. Have you observed any etiological connection between defective drainage of sewers and the disease under consideration?
10. What is your opinion on the pathogeny of the disease? Is it primarily a general disease with consecutive hepatic localisation, or is it an infectious malady of the biliary passages with subsequent general phenomena? What, according to your observations, is the order of the various symptoms (icterus, cardiac symptoms, renal symptoms, &c.)?

I shall be very grateful to all medical colleagues who will communicate their ideas on the above subject to me.

[We have thought it advisable to allow the nomenclature employed in the original French to remain. At the same time we believe "*typhus bilieux*" to be the equivalent of the English term "biliary typhoid."—Ed. J. T. M.]

News and Notes.

TO RELIEVE PRURITUS ANI.—Heat applied to the anus or to other parts of the body affected by pruritus in either a dry or moist form is efficacious in procuring relief. Bathing with very hot water may be the only means at hand; if so, it will be found that water at a temperature of 125° F. is usually capable of being borne for a short time.

Dr. Edmund Andrews of Chicago employs a metal suppository for introduction into the anus for a couple of inches through which hot water circulates, thereby bringing dry heat to bear on the itching part.

MALARIA AT ISMAILIA, EGYPT.—The President of the Suez Canal Company has requested the Liverpool School of Tropical Medicine to send Major Ronald Ross, C.B., F.R.S., to Ismailia, to combat malaria there. Sir Alfred Jones, the Chairman of the school, has agreed to do so, and it is proposed that Major Ross proceed to Ismailia in September.

CANCER RESEARCH.—The Executive Committee appointed to control the fund collected for the purpose of conducting research into the true causes and cure of cancer, consists of Sir W. Broadbent, Sir W. Church, Sir H. Howse, Drs. Sydney Martin, Pye-Smith and Rose Bradford, Professor Sims Woodhead, and Messrs. Langton, Henry Morris, Butlin, McFadyean, and Watson Cheyne.

The fund available at the moment amounts to £32,391.

We would urge upon medical practitioners in the tropics to forward their experiences concerning cancer amongst native races, and we propose to issue a schedule to facilitate reports on this important subject.

Current Literature.

TWO CASES OF THE PINK VARIETY OF MYCETOMA.

By FRANK COLE MADDEN, M.B., F.R.C.S.

Senior Surgeon to the Hospital.

(Reprinted from the "*Records of the Egyptian Government School of Medicine*," 1901.)

SINCE the pathology of madura foot was first placed upon a scientific basis by the investigations of Vandyke Carter, cases of this disease have been described in so many tropical countries that the original idea, that the condition was indigenous to certain districts of India, has gradually been abandoned. Two cases appearing within a year seem to indicate that myce-

toma is not altogether rare in Egypt, and the occurrence of the unusual pink variety of the fungus makes these especially interesting.

A good deal of the pathological obscurity has been removed by many excellent series of cultivation experiments, but still much remains to be done to determine the exact relationship of mycetoma to its more familiar kinsman, actinomycosis. Then, too, the cause of the variation in colour of the mycotic granules, without any marked clinical differences, has still to be explained.

1.—A CASE OF MYCETOMA OF THE RIGHT FOOT.

The patient was a healthy young man, a native of the Soudan, with a very black skin. He described his condition as starting two years ago as a small, hard lump under the ball of the great toe; this lump gradually increased in size, and burst, leaving a small discharging sinus. Similar lumps began to appear on the dorsum of the foot, over the instep, and on the inner side of the sole. The foot gradually enlarged in size in all dimensions, and he had more and more difficulty in walking owing to the increasing pain and weight of the foot. He had a good deal of pain throughout, and for a month prior to admission to hospital, in October, 1898, he had been quite unable to walk. The foot presented all the usual characteristics of a madura foot, and was associated with marked wasting of the muscles of the limb, especially below the knee. On closer inspection of the discharge, with which the innumerable sinuses were tipped, fine carmine-coloured grains were seen sprinkled, as it were, upon the peculiar purulent material which was exuding. Microscopically, these grains were distinctly pink in colour, and under the low power appeared as star-shaped amorphous masses. Dr. Bitter subsequently confirmed the diagnosis of mycetoma microscopically.

As the pain continued severe the patient readily gave his consent to amputation, which was performed by lateral skin flaps in the middle of the leg. The cut surfaces of the bones appeared to be quite free from any mycotic infection.

On cutting into the specimen the bones of the foot were found to be in a condition of gelatinous degeneration, and were hardly distinguishable from the surrounding soft tissue. The rest of the foot consisted of soft, fibrous-looking tissue, and the whole cut surface was scattered throughout with very minute carmine-coloured grains. Even now, when a fresh section is cut, the grains are still bright in colour and widely distributed throughout the section. The general appearance of the specimen did not at all correspond with that of an ordinary madura foot of the white or yellow variety, the absence of cavitation being especially marked.

2.—A CASE OF MYCETOMA OF THE UPPER PART OF THE THIGH.

This man was also a Soudanese, aged 35, born in Darfur. He was living at Kassala when the first symptoms developed a year ago. He noticed a small localised swelling high up on the inner side of the right thigh. This gradually increased in size and extended outwards, its progress being marked by some pricking pain. Three months ago the swelling burst

and discharged a fluid like blood by numerous small openings, a small vesicle forming first at the side of each sinus. The number of openings of this surface of the leg gradually increased, and the whole limb became swollen. The pain has been persistent throughout, though never very severe, and the swelling and oedema became very marked after much walking.

On examination there is a general swelling of the right leg with a well-marked thickening around the upper part of the thigh. Just below Poupart's ligament, extending across nearly the whole anterior surface of the thigh and about a handsbreadth in depth, is the diseased area which forms a distinct elevation above the general swelling. Scattered throughout the area are numerous mammillated tubercles each pierced by a small sinus. Most of the openings are capped by dark red grains, while others are discharging a fluid which is thick with pinkish-brown (terra-cotta) granules. A probe can be passed for some distance into these sinuses and provokes some venous oozing and a profuse discharge of granules. There is considerable oedema all round the area, extending up on to the abdomen and down to the knee, but there is no tenderness. Some deep glands can be felt in the right iliac fossa, though the disease does not appear to extend on to the skin of the abdominal wall.

After remaining *in statu quo* for nearly a month a large abscess formed in the abdominal wall and another smaller one in the thigh. At the operation which necessarily followed incisions were made into these abscesses and a lot of blood-stained purulent material was evacuated. The upper abscess communicated with a cavity underlying the sinus area. An incision was then made through the mass of growth and a large quantity of dirty fleshy tissue was removed with a sharp spoon. The anterior crural nerve was laid bare and masses of growth surrounded the great vessels and extended between the muscles in this region. A piece of a large nerve which had been eaten into and destroyed by the growth was found among the *débris*, which consisted mainly of lumps of sarcoma-like tissue. The cavities were drained and plugged, but very soon further large abscesses formed all down the thigh, and a few weeks later the man died rather suddenly, probably from septic absorption.

A *post-mortem* examination was made by Dr. Symmers, who found "in the right groin numerous fistulous openings communicating with an extensive subcutaneous cavity which was greenish-black in colour and showed points of blue pus and numerous red points of the red form of mycetoma. The bone was unaffected, and the mycetoma seemed to have limited its destructive effect to the skin, subcutaneous areolar tissue, and intermuscular connective tissue, leaving bone and muscles quite free." These *post-mortem* notes are interesting as showing the rather extraordinary local destruction by the mycetoma. One would have expected to find a much more extensive disintegration of the underlying structures after such a long history.

[NOTE.—I had expected to include an account of the pathological and microscopical characteristics of

these cases by Dr. Bitter, but unfortunately he has not had time to complete his investigations.

PLAGUE.

PREVALENCE OF THE DISEASE.

INDIA.—During the two weeks ending June 28th and July 5th, the number of deaths from plague in India numbered 1,684 and 1,058 respectively.

During the two weeks in question the deaths from plague in the Punjab numbered 1,026 during the former week and but 110 during the latter.

In Bombay city, during the two weeks, the number of plague deaths amounted to 48 and 29 respectively.

During the week ending July 5th, the deaths from plague in the Bombay Presidency numbered 565; in Mysore State 178; in Karachi, 43.

In Port Elizabeth, during the weeks ending June 28th and July 5th, the fresh cases of plague numbered 2 and 2, and the deaths from the disease 4 and 2 respectively.

EGYPT.—During the two weeks ending July 13th and July 20th, the fresh cases of plague in Egypt numbered 9 and 15, and the deaths from the disease 6 and 7 respectively. The majority of cases occurred in Alexandria; but at Samalout, Toukh and Daman-hour isolated cases have been reported.

HONG KONG.—During the week ending July 12th, 28 fresh cases of plague and 27 deaths from the disease occurred in Hong Kong, and during the week ending July 19th, 21 fresh cases and 20 deaths.

ODESSA.—Up to July 29th, 7 cases of plague have occurred in Odessa, with 1 death from the disease.

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The Journal of Tropical Medicine.

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Original Communications.

FILARIASIS AMONGST THE BAGANDA.

By J. HOWARD COOK, M.S., M.B.(Lond.), F.R.C.S.(Eng.)

[A map of Uganda will be found in the JOURNAL OF TROPICAL MEDICINE, Oct. 1, 1901.—ED.]

C.M.S. Mengo, Uganda Protectorate.

June 27, 1902.

To the Editor of the JOURNAL OF TROPICAL MEDICINE.

DEAR SIR,—I venture to send you for publication a brief paper on the question of filariasis amongst the Baganda. Many of us have been watching with keen interest, and not a little apprehension, the spread of the terrible malady of sleeping sickness in this Protectorate. The first cases that we saw were reported in the JOURNAL OF TROPICAL MEDICINE for July 15th, 1901. They were seen here in February, 1901. Since that time only sixteen months have elapsed, and now we hear that 10,000 deaths have occurred from this disease. Previous to February, 1901, I never saw a case of *Filaria perstans* amongst the Baganda, though I have examined the blood of very many natives both in films and in the fresh. My present investigations show that 55 per cent. of the natives around Mengo show the filaria in their blood. Do not these figures speak for themselves as to the fatal rapidity of the spread of this incurable disease? I have made an attempt to show on what ground I consider that *Filaria perstans* is connected with sleeping sickness. The numbers of people examined (235) is too small to dogmatise upon, and my conjectures may be wrong, but I think any contribution on so important a question ought to be made public, if only to be substantiated or overthrown by more competent observers. We have hailed with great delight the news that a commission of scientific men is to come out to Uganda to investigate the question, and we wish them every success.

I remain Sir,

Yours faithfully,

J. HOWARD COOK.

THE accompanying statistics represent the result of investigations carried on amongst the Baganda around Mengo with a view to determining the percentage of the inhabitants whose blood is infected with the embryonic form of *Filaria perstans*. A more

limited number of Europeans have also been examined. Lack of time has prevented the examination of larger numbers, and though a statistic built up on the examination of small numbers is misleading, it is hoped that taken in conjunction with similar investigations of other men, it may help towards contributing a useful series of data from which more important deductions may be drawn. An attempt is also made to show how far filariasis, *per se*, contributes clinically detectable symptoms and signs.

Four classes have been examined, members of each class living under more or less similar hygienic conditions.

Class A.—Patients or their friends attending the Out-Patient Department of the Mengo Hospital. These are people all belonging to the "Mukopi" or peasant class. They live in small, round grass houses like beehives, with only a small door and no windows, situated in the midst of the banana plantations. Their food is almost entirely mashed plantain. Their clothing for the most part is a simple barkcloth. They eat out of a common dish, but are most particular about washing their hands before and after food. The food is always well boiled, usually for at least an hour. They sleep often on the ground, and many inmates may be crowded into a small house. For the most part these people were either suffering from minor ailments, coughs, colds, ulcers, &c., or had nothing the matter with them, having only come to fetch medicine. One hundred were selected quite indiscriminately, the only question asked being if they lived in the immediate neighbourhood of the Capital.

Class B.—In-Patients in the Mengo Hospital. These may be taken to represent people who have serious intercurrent disease, or who have undergone surgical operations of the graver type. In each case their disease or operation is indicated, as allowance must be made in the symptomatology for symptoms due to the intercurrent affection, and not referable to the filariasis. Fifty of this class have been examined.

Class C.—Chiefs, boys, girls and women, serving Europeans. These have been included in a separate class, as they live in better houses—more of the European type—feed on more European food, are better clothed, and less herded together. Seventy of this class were examined.

Class D.—Europeans. Fifteen only have been examined.

METHOD OF INVESTIGATION.

In almost every case at least two preparations of freshly-drawn blood were examined. Slides and cover-slips having previously been cleaned with water and absolute alcohol, and the patient's finger-tip having been cleansed with soap and water and then absolute alcohol, a large drop of blood was transferred to a cover-slip, which was then inverted on the slide in the usual way; a second charged cover-slip was then placed by the side of the first on the same slide, and in some cases a third, the finger being wiped dry between the squeezing out of consecutive drops. In this way it was possible rapidly to search two or three preparations on the same slide. A two-third-inch objective was used, and the whole drop of blood carefully and systematically searched in parallel meridians, and the number of filariæ met with in each preparation noted. All the investigations were conducted between 9 a.m. and 6 p.m. This fact and the observed locomotion of the filariæ was taken as working evidence that they were *Filaria perstans*, but actual measurements of the worms was not undertaken from want of apparatus. In many of the cases dried and stained films of the blood were examined by the simple fuchsin method recommended by Manson. But it was noted that the latter method, though so simple and convenient, was not so delicate a test of the presence of filariæ as the examination of freshly-prepared drops of blood, where the filariæ were present in only small quantities, possibly for no other reason than that the movement of the filaria at once arrests attention, and a larger surface has to be searched in scrutinising the films.

In addition to examination of the blood, and prior to it, every person was questioned as to the presence or absence of several of the more commonly occurring symptoms or signs of the earlier stages of sleeping sickness, none of which are, of course, diagnostic of the disease, but all of which, if present, form a suspicious and suggestive clinical picture, whereby one may apprehend the onset, threatened or actual, of the disease. These symptoms have been arranged in parallel columns, a plus sign indicating the presence and a minus sign the absence of the symptom in question. Leading questions were, as far as possible, avoided, or ascertained from the patient's friends. The symptoms and signs sought for were as follows:—

(1) *Tremor of Tongue.*—A fairly constant sign in early stages of sleeping sickness, in my experience, but of course not confined to that condition. In later stages it is accompanied by tremors of lips and other facial muscles, and later still of the skeletal muscles. Probably a sign of debility, but any one who has seen the typical tremor in sleeping sickness never forgets to look at the tongue in suspected cases.

(2) *Enlarged Glands in the Neck.*—Specially a chain

of small shotty glands following the posterior border of the sterno-mastoid muscle.

(3) *Pain.*—Nearly every case of incipient sleeping sickness is, to my belief, accompanied by pain, often of an indefinite character, most frequently in the head and chest.

(4) *Itching.*—Present in a certain number of cases, and in early stages before the characteristic roughness of skin and eruption of later stages makes its appearance. Not always present.

(5) *Facial Aspect.*—In early stages the onset of drowsiness is often suggested by a listless, heavy expression, easier detected than described, and most readily detected in people with whom one is familiar.

(6) *Puffiness about the Eyes.*—Not, of course, diagnostic of the condition. Often present in the latter part of the earlier stages, and, taken with the last sign, contributes to the general dull appearance of the sufferer.

(7, 8) *Loss of Flesh and Strength.*—Curiously enough, not always concomitant signs, as according to their own accounts at all events, some patients grow thinner without losing their wonted energy and strength and others grow weaker without getting thinner.

(9) *Giddiness.*—A very common complaint in incipient cases, and occurs often synchronously with the pain in the head.

(10) *Drowsiness.*—By this is meant rather the mental lethargy of earlier stages than the profound somnolence of the established disease from which the name is derived.

These ten signs and symptoms were sought for, not that they are the only symptoms found in the early stages of the disease, still less that they are in any way diagnostic, but their presence, if marked, in cases that have the filariæ, is suggestive of the likelihood of filariasis running on to the established disease.

Without further preliminary remarks we may proceed to an analysis of the statistics derived from the four classes above mentioned.

Class A.—Peasant class, suffering from trivial ailments, or in good health: 100 cases examined, fifty-five male and forty-five females. Of the total number, fifty-five (55 per cent.) were found to contain the *Filaria perstans*. Of these twenty-seven were males and twenty-eight females. In other words, of the fifty-five males 49 per cent. were filariated, and of the forty-five females 62 per cent. were filariated. The incidence was therefore greater on the females, though very likely this may be corrected by a collation of larger numbers. Amongst the fifty-five who were filariated thirty-six (65 per cent.) had the tongue tremor, while only seventeen (38 per cent.) of the forty-five non-filariated showed this symptom. Amongst the filariated thirty-eight (69 per cent.) had glands in the neck, whilst among the non-filariated only twenty-five (55 per cent.). Among the filariated thirty-nine (71 per cent.) suffered from pain not attributable to other obvious causes, whilst the number of non-filariated with this symptom was twenty-six (58 per cent.). Itching was complained of by thirteen (24 per cent.) of the filariated, and by ten (22 per cent.) of the non-filariated.

A heavy appearance was noted in eighteen (33 per cent.) of the filariated, and in nine (20 per cent.) of the non-filariated. There was puffiness about the

upper part of the face in eight (15 per cent.) of the filariated, and in six (13 per cent.) of the non-filariated. There was loss of strength in twenty-two (40 per cent.) of the filariated, and in fourteen (31 per cent.) of the non-filariated. There was loss of flesh visible in twenty-four (44 per cent.) of the filariated, and in fourteen (31 per cent.) of the non-filariated. Giddiness was complained of in twenty of the filariated (37 per cent.), and in seventeen (38 per cent.) of the non-filariated. Drowsiness was present in eight (15 per cent.) of the filariated, and in five (11 per cent.) of the non-filariated. Now, bearing in mind the fact that after testing their bloods one can hardly over-estimate the number of filariated individuals in a given crowd, but that it is quite possible, and even likely, that an individual whose peripheral circulation is only slightly infected with filariæ may escape detection, especially where so few films are necessarily taken, we may interpret the above results as proving that filariated individuals, even when symptoms of drowsiness have not become a marked feature (15 per cent. as compared with 11 per cent.), yet show symptoms Nos. 1, 2, 3, 5, 8 to a more marked degree than non-filariated individuals; whilst the other symptoms alluded to were more evenly distributed between the two groups, and giddiness was actually more marked among the non-filariated. It must be remembered that these out-patients often complain of giddiness of gastric origin.

The number of filariæ found in a given specimen varied from one to as many as fifty. I have found no relation between the number of filariæ in the peripheral blood and the severity of the somnolence; indeed, in many cases I have seen a disappearance of the worm from the peripheral blood shortly before the onset of a fatal coma.

Turning next to the consideration of *Class B*, the in-patients of the hospital, we notice that out of fifty patients examined twenty-four were filariated—a proportion of 48 per cent. This is a somewhat lower rate than was found amongst the out-patient class, but only slightly so, inasmuch as both were drawn from people of the peasant class. Residence in hospital could not, of course, shield them from filaria infection, and many of them had only recently been admitted. But the interest of these figures lies rather in proving that the hospital is not a focus of infection, but that its inmates actually show a somewhat lower percentage of filariasis than out-patients do. Attention is drawn to this point, as before sleeping sickness was present in epidemic form the earlier cases were admitted as in-patients with a view to clinical observation and study. And occasionally, even with the greatest care, cases find their way in, in the earliest stages, suffering, it may be, also from some other intercurrent disease. Sleeping under the same roof with patients who have sleeping sickness is not unnaturally regarded as a strong predisposing cause of the disease, and, therefore, whatever theory one may have of the mode of transference of the infection, whether by blood-sucking insects or otherwise, yet the obvious precaution is to as far as possible segregate the sufferers and not let them come into contact or sleep under the same roof with the uninfected. In the Mengo Hospital, as above stated, sleeping sickness patients have in the

earlier days of the disease, as found in Uganda, been admitted as in-patients until their death, and even now they occasionally, in the earlier stages, find their way in, in spite of precautions to the contrary. Hence it is with some relief that I found a lower infection-rate in our wards. Moreover, one patient whose hip-joint I had excised last year has been an in-patient for more than twelve months, and yet repeated examinations of his blood have shown no filariæ, though the patient in the next bed to him had six in one drop.

With regard to the comparison of the ten symptoms and signs detailed above as found among the filariated and non-filariated respectively in this class we note as follows: (1) Tongue tremor found in nineteen (79 per cent.) of the filariated, and in sixteen (61 per cent.) of the non-filariated twenty-six. (2) Glands in the neck were found in sixteen (67 per cent.) of the filariated, and in nine (35 per cent.) of the non-filariated. (3) Pain, not distinctly referable to such causes as caused patient to be admitted to hospital, viz., fever, surgical conditions, &c., was found in all of the filariated, a percentage of 45 per cent., and among the non-filariated six (23 per cent.). Itching was found in three (13 per cent.) of the filariated, and in three (11 per cent.) of the non-filariated. (5) A heavy appearance was noted in seven (29 per cent.) of the filariated, and in six (23 per cent.) of the non-filariated. (6) Puffiness about the face in three (13 per cent.) of the filariated, and in two (8 per cent.) of the non-filariated. (7) Loss of strength fell heavier on the non-filariated; thus, filariated six (25 per cent.), non-filariated nine (34 per cent.). (8) Loss of flesh: filariated six (25 per cent.), non-filariated ten (38 per cent.). (9) Giddiness affected two (8 per cent.) of the filariated and four (15 per cent.) of the non-filariated. Lastly, drowsiness was found in five (21 per cent.) of the filariated, but in only one (4 per cent.) of the non-filariated.

Thus we find a relatively higher proportion of the symptoms Nos. 1, 2, 3, 4, 5, 6, 10, whilst 7, 8, and 9, were more marked in the non-filariated. These symptoms, however, viz., loss of strength, loss of flesh, and giddiness are common enough amongst the type of people found in hospital wards.

We next turn to *Class C*, the chiefs and natives living in the service of Europeans. Of this class seventy were examined and of that number twenty-five (36 per cent.) were filariated. This is a distinctly lower proportion than was found to obtain among the peasant class.

Tongue tremor was found in seventeen (68 per cent.) of the filariated, and in twenty (44 per cent.) of the non-filariated. Glands in neck found in sixteen (64 per cent.) of the filariated, and in sixteen (35 per cent.) of the non-filariated. Pain was found in nine (36 per cent.) of the filariated, and in fourteen (31 per cent.) of the non-filariated. Itching was found in four (16 per cent.) of the filariated, and in five (11 per cent.) of the non-filariated. A heavy appearance was noted about seven of the filariated (28 per cent.), and in thirteen (29 per cent.) of the non-filariated. Puffiness about the face was noted in two (8 per cent.) of the filariated, and not seen in the non-filariated. Loss of strength was found in three (12 per cent.) of the filariated, and not found in

the non-filariated. Loss of flesh was found in two (8 per cent.) of the filariated, and in three (6 per cent.) of the non-filariated. Giddiness was found in four (12 per cent.) of the filariated, and in three (6 per cent.) of the non-filariated. Lastly, drowsiness was found in one (4 per cent.) of the filariated, and not found in the non-filariated.

When we come to *Class D*, the Europeans in Mengo, we do not find the filariæ. Hence a comparison between the two classes is no longer possible.

The results of the preceding paragraphs may thus be summarised in tabular form:—

| | <i>Class A.</i> | | <i>Class B.</i> | | <i>Class C.</i> | |
|---------------------|-----------------|------------------|-----------------|------------------|-----------------|------------------|
| | % in Fil. | % in Non-fil. | % in Fil. | % in Non-fil. | % in Fil. | % in Non-fil. |
| Tongue Tremor .. | 65 | 38 | 79 | 61 | 68 | 44 |
| Glands in Neck .. | 69 | 55 | 67 | 35 | 64 | 35 |
| Pain .. | 71 | 58 | 45 | 23 | 36 | 31 |
| Itching .. | 24 | 22 | 13 | 11 | 16 | 11 |
| Heavy Appearance .. | 33 | 20 | 29 | 23 | 28 | 29 |
| Puffy .. | 15 | 13 | 13 | 8 | 8 | 0 |
| Loss of Strength .. | 40 | 31 | 25 | 34 | 12 | 0 |
| " " Flesh .. | 44 | 31 | 25 | 38 | 8 | 6 |
| Giddy .. | 37 | 38 | 8 | 15 | 12 | 6 |
| Drowsy .. | 15 | 11 | 21 | 4 | 4 | 0 |

These percentages are reckoned, of course, not with regard to the total number of patients examined, but separately in each group, relatively to the total number of filariated and non-filariated individuals of each class.

CONCLUSIONS.

(1) That a high proportion of the Baganda living in the immediate neighbourhood of the capital, Mengo, harbour the embryos of the parasite *Filaria perstans* in their blood.

(2) That different classes of natives show varying infection-rates; thus the peasant class were most affected (54 per cent.), then the sick in hospital (48 per cent.), and those who lived in the best hygienic surroundings fared best, chiefs and Europeans' servants (36 per cent.).

(3) Some cases of filariasis occur with absolutely no further symptoms of disease. Hence, in all probability they act as an immense source of danger to the uninfected population, and owing to the insidious onset of early symptoms, any attempt at segregation of filariated individuals must be attended with almost insuperable difficulties.

(4) That the majority of cases of filariasis are accompanied by some at least of the earlier symptoms of sleeping sickness, and hence a connection between the two conditions is highly probably. Especially if the converse be considered, viz., that a very high proportion of cases of sleeping sickness, if examined at the right stage, readily show the filaria in the peripheral blood stream—thirty-two cases out of thirty-four examined in the Mengo hospital.

(5) Although I have not found that the severity of somnolence depends on the number of *Filaria perstans* in the blood. I think that, as a rule, the number and severity of the earlier symptoms do vary with the number of the embryos in the blood.

(6) Some other factor, in addition to the presence of filariæ in the blood, is required to aid the diffusion both of filariasis and of sleeping sickness, otherwise the hospital in which cases both of filariasis and occasionally sleeping sickness have been introduced ought

to act as a centre of infection, which it appears not to have done.

(7) Experiments ought to be carried out to determine what this factor is, e.g., testing the various blood-sucking insects on filariated individuals, to determine if they cause diffusion of filariasis, cf., *Filaria nocturna* and *Culex*; also, if possible, to determine how filariasis oversteps the boundary line that borders on the established disease of sleeping sickness.

Class A.—PATIENTS ATTENDING OUT-PATIENTS' DEPARTMENT MENGO HOSPITAL, C.M.S.

One Hundred Patients.

Sex: M., 55; F., 45. Tongue, 53. Glands, 63. Pains in—chest, 20; head, 12; abdomen, 12; head and body, 4; head and chest, 7; body, 8; dyst., 1; chest and abdomen, 2. Itching, 23. Heavy appearance, 27. Puffy, 14. Loss of strength, 36. Loss of flesh, 38. Giddy, 37. Drowsy, 13. *Filaria perstans*, 55 per cent.; number of filariæ, 1–50. Slides used, 2 or 3. Address: Mengo, 58; Makerere, 6; Namirembe, 3; Mumgongo, 2; Rubaga, 2; Bira, 2; various other localities, 27.

Class B.—PATIENTS RESIDENT IN C.M.S. HOSPITAL, NAMIREMBI.

Fifty Patients.

Sex: M., 31 (filariated, 14); F., 19 (filariated, 10). Tongue, 35. Glands, 25. Pains in—head, 7; chest, 3; body, 1; abdomen, 4; head and abdomen, 1; head and chest, 1. Itching, 6. Heavy appearance, 13. Puffy, 5. Loss of strength, 15. Loss of flesh, 16. Giddy, 6. Drowsy, 6. *Filaria perstans*, 48 per cent. Slides used, mostly 2.

Class C.—CHIEFS AND EUROPEANS' SERVANTS.

Seventy Patients.

Sex: M., 67; F., 3. Tongue, 37. Glands, 32. Pains in—head, 5; chest, 7; chest and body, 1; abdomen, 7; chest and abdomen, 1; head and chest, 1; back, 1. Itching, 9. Heavy appearance, 20. Puffy, 2. Loss of strength, 3. Loss of flesh, 5. Giddy, 7. Drowsy, 1. *Filaria perstans*, 36 per cent. Slides used, mostly 2. Address: Namirembe, 36; Hospital boys, 10; Mengo, 10; Bulange, 12; Kampala, 1; Ndeje, 1.

Class D.—EUROPEANS.

Fifteen Persons Examined.

Sex: M., 9; F., 6. Tongue, 4. Glands, 1. Drowsy, 2. Slides, mostly 2 to 4. Address: Namirembe, 8; Ndeje, 5; Bulange, 1; Ntebbe, 1.

BERI-BERI AT DIÉGO GARCIA.

By Dr. J. BOLTON.

Sanitary Warden.

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DIÉGO GARCIA, the most southern island of the Chagos group, is situated 7° S. and 72° E. It is a narrow sand-bank, thirty miles long, shaped like a horseshoe; in breadth it varies from a few feet to two miles and more. The horseshoe encloses a magnificent bay, fifteen miles long and over five miles wide at certain parts.

There is no soil properly speaking at Diégo. The whole island is composed of a layer of sea sand of various thicknesses reposing upon a bed of madreporic formation. Here and there, where the brushwood is thick, a certain quantity of vegetable loam derived from decayed leaves is found mixed with the sand, to which it imparts a dark grey colouring. Vegetation is most luxuriant, and some forest trees attain large dimensions.

The whole island is reef-bound except at two or three places in the bay.

It is almost entirely covered with cocoa-nut plantations of different ages. Here and there may be seen clumps of forest trees of great height and size. A belt of low trees grows all along the coast on both sides of the island.

There are two oil factories at Diégo; one at Pointe de l'Est, the most important, and the second at Pointe Marianne.

The population of the island, composed of 466 souls, is divided between the two stations in the following proportion: 326 at the former and 140 at the latter.

AN OUTBREAK OF BERI-BERI.

The unusually high mortality observed this year is due to an epidemic of beri-beri, which began at Pointe de l'Est during the latter part of last and prevailed during the first half of this year.

The origin and progress of the epidemic were, as far as I could gather from the books of the estate and from information supplied me by Mr. de Caila, the Manager, as follows:—

On July 27th, 1900, eight men and one woman, Mourima, Hosseinee, Vitah, Sambetti, Alfany, Moussa, Allee, Toolee, Sapikalee (w), landed at Diégo Garcia from Mauritius.

Although recruited in Mauritius, they had shortly before arrived from *Glorieuse*—they were natives of the Comores islands and of the East African coast.

When they landed at Diégo Garcia, Mr. de Caila was struck with their appearance. Seven had sore eyes, sore mouth and gums and swollen feet. Alfany suffered such severe pains in the legs that he could hardly walk.

Three days after their arrival Hosseinee and Mourima applied for hospital treatment. The former had painful swollen legs, epigastric distension, sore gums. No fever. The swelling of the legs steadily progressed upwards as far as the knees. He kept going backwards and forwards to the hospital till January 14th, 1901, when he showed signs of cardiac distress, he breathed with difficulty, had palpitation, irregular pulse; he had also œdema over the sternum, and the pit of the stomach was very prominent. He died suddenly next day, January 15th, 1901.

In Mourima's case the symptoms were mild: pains in legs, œdema of ankles and calves. No cardiac distress. Recovered spontaneously at the end of a fortnight after having taken a couple of purgatives.

On August 6th, their friend, Vitah, was sent to hospital for swollen legs, pain in the calves and purulent (?) ophthalmia.

He refused to remain, but applied frequently as an out-patient for treatment.

In the beginning of the month of January, 1901, he applied for admission as he felt worse. Mr. de Caila then noticed great swelling of the legs, which were also painful and tender, distension of the epigastrium, œdema over the sternum, great cardiac distress, with irregular weak pulse. No fever. He died suddenly the 12th of the same month.

The fourth case which came under observation was that of Sambetti, who showed the first symptoms in August last year. He used to attend the hospital

occasionally. The œdema of the legs gradually disappeared, and paralysis of the legs remained. On June 11th he was sent back to Mauritius, where he was admitted a patient of the Civil Hospital.

The next case among these Johannese was observed upon Alfany, who showed swollen and painful lower extremities; slight cardiac distress. He was sent back to Mauritius, and is reported to have got well on the voyage.

Allee, Moussa, Toolee, and the woman Sapikalee did not present any symptoms beyond stomatitis and ophthalmia.

Up to now the disease had been limited to the Johanna gang. On March 27th, Louis Némorin Celestin, the hospital attendant, who, although he did not live anywhere near, or in the same camp as the Johannese labourers, had been, however, in constant attendance upon them, was found suffering from what he thought was colic. Mr. de Caila, however, at once notified that the abdomen and legs were swollen, and the latter tender on pressure. On April 8th, feeling, as he thought, better, he left the hospital against his master's advice. Next day he was brought back with violent pains over the heart, difficulty of breathing, præcordial œdema, and increased swelling of abdomen and legs.

He died suddenly at noon that day, although his condition in the morning did not indicate the possibility of such a sudden dissolution. A few minutes before death he had vomited some bloody liquid. He used to remain in the huts of the Johanna men several hours at a time attending upon them.

On March 25th, Albert Sylvestre Gaïqui was admitted into hospital. For nearly one month previously he been complaining of numbness, tingling and pains in his hands, calves and feet. He gradually developed swelling of feet and legs, soon to be followed by sternal œdema and cardiac distress. Towards the end the face was slightly puffy.

The swelling got better under treatment (purgatives), but the cardiac distress persisted, and he died suddenly on April 28th.

On March 15th Jean Baptiste Valentin sickened with same symptoms and died on April 29th.

On May 9th a man named Joseph Wilson, a Malegasy, who, although he did not live near, nor in the same camp as the Johanna men, used to camp out with some of them (Alfany, Moussa, Allee and Sambetti) complained of nausea, palpitation, "pain in the heart." He had œdema over the sternum. No fever. He died suddenly next day. Five minutes before death he was walking in the ward.

The next person to be attacked was an Indian woman, named Edmée Verasamy. She had been confined on May 10th. A few days after, when in apparent health, she complained of pains in the legs. Mr. de Caila noticed the legs and the abdomen were swollen; cardiac distress was also observed. She gradually sank, evidently from heart failure, and died on May 22nd. She was conscious to the last minute.

On June 11th her husband, A. Verasamy, showed symptoms of Beri-beri, and died five days after. He was walking about in the ward, when he was seized with cardiac distress, and was dead in a few minutes.

Shortly before her death, Edmée had asked Mrs. de

Caila to adopt her baby. This she did and took it to her house on May 22nd. About the middle of June he showed symptoms of beri-beri and died on the 23rd of the same month.

Jules Imouche, who was the godfather of Armand Sylvestre Gaïqui, attended upon him during his illness and used to sit up all night with him. He developed symptoms of the disease shortly after his godson's death; his wife and son soon after him complained of similar phenomena. They all recovered. When I landed at Diégo I saw Jules Imouche. He had well-marked atrophy, with slight paralysis of the right leg. The left felt heavy. Besides the above, the following had undoubted symptoms of the disease in a more or less mild form, viz.: Arthur Francis, Jean Michel, and Joseph Moolati. *The first used to camp out with the same men as Wilson*; the second nursed J. B. Valentin; the third had watched over the dead body of L. Némorin on April 11th. About ten days after he noticed his feet were swollen; he, however, continued to work till May 5th, when he remained one day in hospital. He was treated at home till the 13th, when he resumed work. He is perfectly well now, although he still complains of a certain heaviness in the legs.

Mrs. de Caila, who had been most untiring in her care of the sick, nearly fell a victim to the disease. She had undoubted symptoms of it. Edema of lower extremities and over sternum; pain in legs. Cardiac distress, which at times was very severe; numbness in legs.

On July 1st *George*, the brother of Verasamy, applied for treatment. He presented symptoms of beri-beri. He recovered, and his was the last case observed.

The treatment adopted was purgatives, tonics, and good food. As the Johanna lot were all Mahomedans they were fed upon eggs, condensed milk, and fowls.

Besides the Johannese labourers, there were many others with them on board. Some were landed at Pointe Marianne, Point de l'Est, and others later on at the island of Peros Banhos. Not a single case of the disease was observed among them.

The Johanna gang was housed in a row of three huts of two rooms each, Nos. 1, 2, 3. The first two cases among them occurred in No. 1, the third and fourth in No. 3, and the fifth in No. 2.

Up to March 27th the disease seemed to have been confined to them. On this day, however, the hospital warder, L. Némorin, who did not live near them, but had been in constant attendance upon and nursing them, showed undoubted symptoms of the disease, and died a fortnight after. A. S. Gaïqui, who died on April 28th, lived in a hut opposite opposite Vitah's, but about 100 feet distant; J. B. Valentin's was behind Vitah's, and Joseph Wilson's was near Némorin's, but far from the others and to the windward.

Jules Imouche and family lived about fifty yards from Sambetti and Vitah's hut and to the windward.

The huts occupied by the Johanna gang had concrete floors in very good condition, except at No. 3, where it was broken up.

These huts have been burnt down.

On June 11th, the remaining Johannese were shipped back to Mauritius. On the 16th G. Verasamy sickened in a hut opposite Vitah's, in which Sambetti lived up to the day of his departure. This was the

last case, and the disease disappeared. No fresh case occurred on board during the passage back, which was a very rapid one (7½ days).

DEATH-RATE AND INFECTIOUS NATURE OF THE DISEASE.

There can be very little doubt that the disease which prevailed at Diégo and killed nine persons out of twenty-one who were attacked was beri-beri of the mixed form, which is not attended with considerable anasarca.

The death-rate was nearly 43 per cent., and the attack-rate on total population of the infected camp was 11.5 per cent.

It is evident that it was imported into the island by the particular gang of labourers who had arrived from *Glorieuse* Island some time before they were shipped for Diégo.

The disease spread over a certain area of the camp round the huts occupied by them.

The infectious nature of the disease was well established in the case of J. Wilson and the hospital attendant. Both these men did not live anywhere near the infected huts; the former, however, used to camp out with some of the sick men, and the latter, from the nature of his duties, was in constant attendance upon the sick, and therefore in contact with them.

The case of Jules Imouche and family is also very instructive. His godson, A. S. Gaïqui, being ill of disease, he brings him to his own house for treatment. He infects the whole family (three persons).

In the case of Mrs. de Caila, there can be no doubt that she caught the disease from the infant son of Edmée Verasamy. This baby had been removed to the main house after its mother's death; it was placed there under very much altered and improved conditions. It had a nice cot in a large, well-aired and lighted room, eight feet above the soil. It was nursed day and night by Mrs. de Caila, but it died, after having infected her. This baby had been removed from the infected area three weeks before it showed any symptoms of the disease, and died after an illness of eight days.

There is ample evidence to show that the disease was conveyed to Diégo by a gang of labourers, and that it was afterwards carried more than five miles away by some of them to an outlying post, where Wilson was infected.

Dr. Manson is of opinion that the germ of the disease "resides in the soil, in the houses and surroundings of beri-beri spots."

The soil is the infected medium; the man residing on it is poisoned, not infected.

The manager's house at Diégo is distant more than half a mile from the infected camp. It is constructed upon a stone foundation eight feet high. The main building itself is well constructed of teak timber.

The house is kept scrupulously clean and well aired. The baby who died in this house had been a resident there for three weeks before it fell ill, and while she was nursing it Mrs. de Caila began to notice the first symptoms of the disease upon herself; the baby had come to know her so well that it had to be kept in her lap to prevent it crying.

It is a significant fact that out of the seven persons sleeping in this house Mrs. de Caila should have been the only one attacked. She was in direct contact with the child. The dispenser did not live anywhere near the infected area.

THE FOOD SUPPLY

(deficient nitrogen theory) cannot in any way be impugned.

The men of Diégo have every chance of nourishing themselves well, and they take advantage of the facilities offered them.

It is true that the Johanna gang were Mahomedans, and as such did not eat pork, but they had every facility for obtaining fowls, eggs, sea-birds, and vegetables of all kinds, and limes are given to the men with a very free hand. On the other hand, the other inhabitants of the island who were attacked by the disease were young, strong, and well nourished, and the rice distributed to all the labourers, both of Pointe Marianne and Pointe de l'Est, had been in store before the arrival of the Johanna men, and was used before their arrival, during their stay, and after their departure without any untoward effect. I do not believe that dampness has anything to do with the production, maintenance or propagation of the disease. 1900 was an exceptionally dry year. When I arrived at Diégo I was told rain had not fallen for more than six months. The huts of the camp occupied by the majority of Beri-beri cases were dry, well ventilated and lighted, and possessed concrete floors. The manager's house is unusually dry and well ventilated.

THE HISTORY OF THE EPIDEMIC SEEMS TO TEACH :

That the germ of the disease, vegetable or animal, may remain latent in the human body for months, until local conditions favourable to its development obtain. It then proliferates and gives rise to the characteristic symptoms. I was told these Johanna men were loathsome in their habits; they were located together with a number of other passengers in the fore part of the "tween deck," which is always ill ventilated, damp, stuffy and dark.

So that, coming from one of the Comores Islands with probably the germs of beri-beri latent in them, they found themselves on board under conditions favourable to the proliferation of the microbe, and landed at Diégo with the disease in a mild form.

Some time afterwards they communicated it to some of their nearest neighbours—to Wilson and to the hospital attendant.

It would also appear as if the Johanna men were, so to speak, the culture media in which successive crops of germs were produced, and thus kept up the infection of the soil or air. The disease disappeared with the departure of these men. Again, although the huts occupied by Verasamy and others have since been reoccupied, no fresh case of the disease has appeared in them.

There are some points of resemblance between the outbreak at Diégo and an epidemic of the disease which occurred in Fiji in 1894.

In that year 250 Japanese coolies were imported for a plantation at Laboosa in the month of April. *Many of them had had the disease previous to their embarkation.*

One month after their landing the first case was observed, that is, in May; from that time up to September only three cases had occurred. The disease then took up an epidemic form. By February, 1895, only 181 remained out of 250, and they were sent back to Japan. The disease disappeared then and has not apparently reappeared.

At Diégo some of the Johanna men were sick on landing in July. It was only in December, *five months after*, that the disease attacked other inhabitants of the camp.

CLIMATIC BUBOES IN KUSAIE, CAROLINE ISLANDS.

By The Rev. C. F. RIFE, M.D.

In a letter addressed to Dr. Manson, dated May 29th, 1902, Dr. Rife writes:—

"Having been for eight years a medical missionary on the island of Kusaie, on the most easterly island of the Carolines, I was much gratified last year to learn while in the United States on furlough, of your book on TROPICAL MEDICINE. I have been much interested in it since my return, as the books of a temperate climate are totally inadequate to a practice here. I have recently seen about thirty cases of climatic bubo, fifteen of which were under my immediate supervision, and as many more who were outside patients, and not seen by me more than once or twice. I have a training school for Marshall Islands natives, and have now twenty-one pupils, young men. About six months ago, four of them and myself made a tour of these islands on a small schooner that had just returned from Guam. One of the sailors had a long attack of climatic bubo, and one of my boys contracted it while we were on board ship. Since coming ashore, all but six of the number have had it. They are rather closely associated in sleeping and bathing. Two of the fifteen think that they had no previous wound for the infection to enter, but I have doubts about this. The others all had some abrasion of the skin to begin with. At about three to five days, apparently, from time of infection, the lower inguinal glands, usually but one, would become much enlarged (as you say, to about the size of a hen's egg), there would be a chill, headache, considerable pain in the back, and some fever. I always painted the surface over the gland with tinct. iodine, and sometimes made a second application. None of these cases suppurated, but I saw three outside cases which it was necessary to lance. The trouble at the seat of infection, however, was not so easily got rid of. There was much suppuration, and in two cases rather extensive burrowing, which required the knife. The ulcers which I had to deal with were not more than an inch in diameter, rather deep, with a punched-out margin, and much congested in the surrounding skin. After a period of suppuration of from one to four weeks, there would be a long and tedious recovery, with an ichorous discharge which lasted for weeks. There was much deep pain, and this seemed out of all proportion to the amount of ulceration. The pain was so severe that they could not stand for any length of

time, and when at their worst it was almost impossible for them to walk. The cases which recovered quickest were well in four weeks, while some were twelve weeks in recovering.

"All the cases in my school were below the knee, one boy also having two small ulcers on one hand. The Kusaians report the case of a woman who had a very large ulcer on the thigh. Of my boys, five had but one ulcer, while one had six. No one had enlarged glands except with the first infection. I found the treatment of the ulcers rather unsatisfactory. Nothing seemed able to abort them in their long course. I tried hot fomentations, plain and medicated, with no apparent success. After the period of suppuration was passed, the pain was much relieved by dusting the ulcers with calomel.

"Our climate is very moist. In 1899 I measured 256 inches of rainfall, and think I will have much more than that this year. Extremes of temperature are 75° and 90° F. Our island is high, about 26 miles in circumference, and has a resident population of 500, with about 120 natives of the Marshall and Gilbert Islands, who are in training schools."

THE GAMBIA MEDICAL EXPEDITION.

THE eighth expedition of the Liverpool School of Tropical Medicine, under Dr. J. F. Dutton and Dr. J. L. Todd, will start for the Gambia and French Senegal on August 23rd. The French Government having been invited to attach a scientist to the expedition, M. Doumergue, the Minister of the Colonies, has written to say he would have been glad to accept the invitation, and to lend his support to an undertaking which is of the utmost importance to the future of colonisation, but that his department has itself just arranged to send to the French West African possessions special doctors for the purpose of studying the different tropical diseases there. He adds, however, that these doctors will be delighted, if occasion offers, to associate themselves with the Liverpool School of Tropical Medicine in its work.

DONATIONS TO THE LIVERPOOL SCHOOL OF TROPICAL MEDICINE.

At the dinner to the members of the Tropical Diseases Section of the British Medical Association at Liverpool on August 1st, the Chairman, Sir Alfred Jones, K.C.M.G., announced that "a good friend of the School had given £25,000 to provide the necessary laboratories and other buildings, and he was proud to be able to announce that owing greatly to the energy and perseverance of Professor Boyce, £10,000 had been contributed for the endowment of a chair, the incumbent of which, he hoped, would never be less worthy than their present professor, Major Ross." We congratulate the Liverpool School of Tropical Medicine on their good fortune.

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THE

Journal of Tropical Medicine

AUGUST 15, 1902.

British Medical Association.

SECTION OF TROPICAL DISEASES.

SECOND NOTICE.

DISCUSSION ON DYSENTERY.

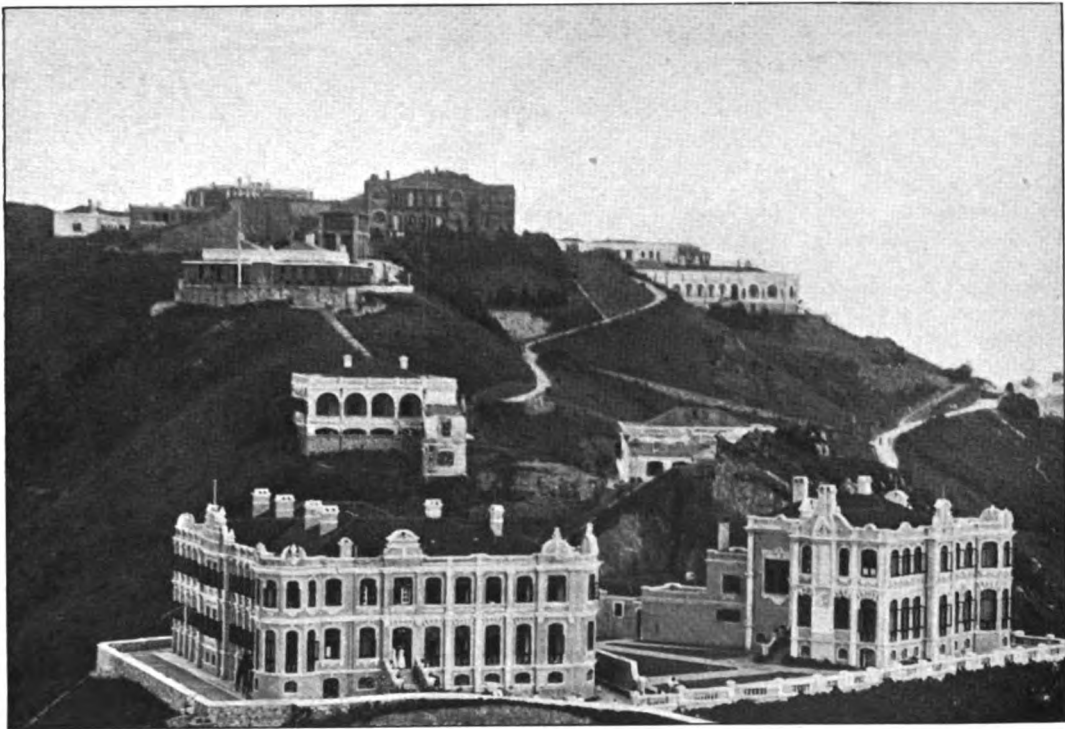
[The papers read will be printed in full in subsequent numbers of the Journal; an abstract of the papers is here appended.]

I.—ANDREW DUNCAN, M.D., B.S.Lond., F.R.C.S., F.R.C.P., Lt.-Col. I.M.S. (retired).

UNDER the term dysentery more than one affection is included; the arguments for this conclusion are based on bacteriological investigations and the results of treatment.

Micro-Organisms.

Of the micro-organisms having a causative effect we have: (1) those of Ogata and Shiga (Japan); (2) Flexner's organism (Philippines); (3) the bacilli coli dysenteriae (Celli and Fiocca); (4) pyogenic cocci; (5) bacillus pyolaxeus (U.S.A.); (6) paramoecium coli in Cochin China (Treille); (7) amœba coli. The arguments advanced to prove that the amœba coli is the potent factor in causing dysentery and liver abscess has, as far as dysentery in India is concerned, not been proven. Hepatic abscess is rarely connected with dysentery in the West Indies, with the asylum



THE PEAK HOSPITAL, HONG KONG, WITH DOCTOR'S RESIDENCE ADJACENT,
RECENTLY BUILT.

dysentery in England, and has scarcely been seen in the thousands of cases of dysentery which occurred during the South African war. The presence of the amoeba in dysentery would appear to be a mere concomitant.

Treatment of Dysentery.

In acute cases in India there seems no sufficient reason to discard ipecacuanha properly administered. Cinnamon, when ipecacuanha cannot be tolerated, seems in drachm doses occasionally a useful substitute. Salines are commended in enthusiastic terms by many medical officers in India and elsewhere. Treatment by the *Monsonia ovata* is advocated by physicians in South Africa. Lavage of the intestine by boracic acid finds supporters, and so also the administration of sulphur and Dover's powder in certain conditions. In the treatment of chronic dysentery, bismuth in 20-grain doses with a minute dose of Dover's powder (gr. $\frac{1}{4}$) is at times attended by satisfactory results; and so also is the administration of castor oil, 2 drachms every second night with a nightly suppository of 2½ grains of liquid extract of opium.

II.—W. J. BUCHANAN, Major I.M.S. "THE PREVENTION AND TREATMENT OF DYSENTERY IN INSTITUTIONS IN THE TROPICS."

Based on an experience of 1,130 consecutive cases of dysentery, with only nine deaths, Major Buchanan states that amoebic dysentery is by no means so common as the bacillary form. Practically the amoebic dysentery is only recognised by the occurrence of liver abscess, the finding of the amoeba, or the absence of reaction with Shiga's bacillus. In the 1,130 cases under consideration, not a single case of liver abscess in the living or dead was found. The dysentery of Indian prisons is probably almost entirely bacillary, and the applied tests have shown it to be due to Shiga's bacillus. Yet although the amoeba and Shiga's bacillus are accepted as the causes of the varieties of dysentery, we are ignorant of the means whereby these organisms are carried into the human body.

Food and Drink as Causes of Dysentery.

Although water is admitted as the medium for the conveyance of typhoid, cholera and dysentery, few will admit that it is the only means of conveyance of these diseases. Specifically polluted water can certainly account for sudden widespread outbreaks of these diseases, but will it equally well account for the steady low persistence of cases after the defects in the water supply have been repaired? In other words, the water theory will explain epidemic prevalence, but it will not so easily explain endemic persistence. With regard to food as a cause of dysentery in institutions, although almost every article of diet has been condemned in turn, the real cause has not been arrived at. Certainly good cooking and good, well-cleaned (grain) warm food are efficient factors in checking dysenteric outbreaks.

Communicability of Dysentery.

So convincing are the proofs that dysentery can be communicated from the sick to the healthy, that it

seems imperative to (1) isolate patients suffering from dysentery; (2) disinfect all clothing &c., used by dysenteric patients; and (3) disinfect and incinerate dysenteric stools.

Treatment.

Curdled milk, boiled milk or rice-water, sago, arrowroot or such starchy substances, give the best results. Castor oil and laudanum to begin with is advisable; salines in acute cases answer well; ipecacuanha is worthy of respect; enemata may be useful; Izal is well-nigh useless; sulphur is not to be trusted.

III.—LEONARD ROGERS, Major I.M.S. "TROPICAL OR AMOEBIC ABSCESS OF THE LIVER AND ITS RELATIONSHIP TO AMOEBIC DYSENTERY."

The constancy of the amoeba in tropical liver abscess is a striking fact in pathology. By tropical liver abscess is meant the large single or multiple abscess which is clinically recognisable as such during life, as opposed to multiple small pyæmic abscesses.

The amoeba is seldom found in the liver pus which first issues on opening an abscess, but it is almost constantly found in scrapings of the liver abscess wall. On the other hand, amoeba may be present and then disappear from the liver pus.

The Frequency of the Association of Dysentery with Liver Abscess.

Neither the clinical history alone, nor the *post-mortem* examination alone, afford correct information as to the frequency of this association, and it is only by examination of a series of cases, in which both clinical and *post-mortem* records are available, that the true proportion of cases in which dysentery is associated with liver abscess can be ascertained. The evidence collected strongly points to dysentery being an invariable accompaniment of the amoebic or tropical liver abscess.

The Value of Leucocytosis in the Diagnosis of Liver Abscess.

In Major Rogers' experience leucocytosis is most marked in comparatively small, deeply-seated abscesses of the liver, and less in those which are already beginning to come to the surface.

The Situation of the Liver Pus.

Major Rogers remarks that he has been frequently struck by the fact that even comparatively small amoebic abscesses are nearly always situated just under the surface of the diaphragm, between that organ and the liver, in the neighbourhood of the suspensory ligament.

Treatment.

Seeing that liver abscess is caused by the amoeba dysenteriae and that quinine in a solution of 1 in 5,000 will kill this organism, it seems rational to apply a solution of quinine to the abscess cavity, in the hope of checking the suppuration. This solution might be applied either by injection after merely aspirating, or as a lotion wherewith to wash out a freely-opened abscess.

DISCUSSION.

PATRICK MANSON, C.M.G., F.R.S., LL.D. (Physician Seamen's Hospital Society), remarked that

there were perhaps half a dozen diseases included under the term dysentery. He wished to convey a word of warning against relying too implicitly on agglutination tests as a scientific basis of differentiation of diseases. The term, amoebic dysentery, is no doubt a "catching" term, but it was just possible that it was not quite accurate. In certain types of dysentery recovery was the rule under any form of rational treatment, but in sloughing dysentery the opposite obtained. The potency of the bacillus of Shiga, and the influence of the amoeba in producing dysentery, has still to be proved.

The cases of dysentery of tropical origin Dr. Manson met with in London were necessarily more or less of a chronic nature. In such cases he had found ipecacuanha a very efficient remedy. Given in large doses at first, and continued in lessening doses daily for a week or ten days, he had obtained excellent results with ipecacuanha. It was usual to administer this drug only in acute cases, but it was evident that if ipecacuanha is potent in destroying the specific agent of dysentery in the acute form of the disease, there was every reason to believe it would do so in the more chronic cases. Rest in bed and the usual "soft" diet were essential principles to be followed in the treatment of the disease, and an occasional dose of castor oil. Salines were not without danger, at any rate in chronic dysentery where ulcers obtained, and the treatment by salines had not proved efficacious in his practice.

The Hon. Dr. W. G. Rockwood (Ceylon) stated that he was of opinion that dysentery was to some extent a communicable disease, and that certain cases assumed a malignant character and proved fatal in three or four days, in spite of all known methods of treatment. He had employed the saline method of treatment with excellent results in the simpler forms of dysentery. He had seen liver abscesses in females, but he looked upon liver abscess in females as being a very rare disease.

Dr. J. H. Musser (Philadelphia, U.S.A.) referred to the work of Flexner in connection with the bacillary form of dysentery due to the bacillus of Shiga. He upheld diagnosis by agglutination tests. Abscess of the liver is not a purely tropical disease, for Dr. Musser had seen isolated cases in persons who dwelt in Philadelphia.

EDWARD HENDERSON, M.D. (late of Shanghai), observed that between the years 1868 and 1875 in Shanghai, dysentery was more severe than during any period of his subsequent practice in China. Dr. Henderson was accustomed to classify the cases he met with into catarrhal and diphtheritic; the former all recovered, the latter nearly all died. Ipecacuanha and other drugs, when administered in cases of dysentery that tend to get well of themselves, get the credit of curing without any justification whatever; ipecacuanha in diphtheritic cases did no good and in some cases seem to do positive harm.

JAMES CANTLIE, M.B., F.R.C.S. (Surgeon Seamen's Hospital, London), believed that in serious cases of dysentery the most effective plan of treatment would prove to be to perform a colotomy in the right lumbar region and wash out the large bowel by frequent douchings of warm boracic acid or sulphate of copper

solutions. He agreed with Major Rogers that the most frequent site of abscesses, in which amoeba were found were in what Mr. Cantlie had termed the supra-hepatic abscesses, as distinct from the intra-hepatic abscess. He had seen several cases of supra-hepatic abscess occur in men who had only been in the tropics a few weeks and who had neither suffered from malaria nor from dysentery, or any intestinal flux that could be considered in any way dysenteric.

Inspector-General TURNBULL, R.N. (retired), said he met with three varieties of dysentery in Hong Kong: (1) True sloughing dysentery; (2) an aphthous dysentery, commencing with aphthae of the mouth and "going through the patient"; and (3) hypertrophy of the walls of the large bowel with erosion of the mucous membrane. He had treated three cases of liver abscess in Hong Kong by Manson's trocar and cannula. All the patients completely recovered and two of them remained in the tropics for many years afterwards without any recurrence of the disease.

F. M. SANDWITH, M.D. (Professor of Medicine, Medical School, Cairo), said the most important question to decide was, what dysentery really is, and how the poison enters the body. Asylum dysentery and ulcerative colitis existing in non-tropical countries are quite different to the typical dysentery of the tropics. The former disease, however, probably also occurs in "institutions" in the tropics and often yields readily to treatment. It is probable there are more forms of dysentery than the two we are accustomed to term the amoebic and the bacillary. He has believed for many years that dysentery is an infectious disease, and should be treated with the same precautions as typhoid. Professor Sandwith ventured to draw a parallel between the possible production of dysentery by infected rice and the terminal dysentery met with in pellagra, a disease known to be caused by diseased maize. Outside India ipecacuanha seemed useless, and he had long renounced the drug in his practice. If it is useful at all it is in chronic dysentery, when it should be given in small frequently repeated doses. Enemata he had long advocated, and he looked upon the practice of washing out the bowel by enemata of boracic acid solution, or of sulphate of copper, as not only the best method of treatment practically, but also as being the rational form of treatment to pursue.

M. LOGAN TAYLOR, M.B., Ch.B. (Glasgow).
Sanitary Work in West Africa.

After describing the state of Freetown and Cape Coast Castle as to their insanitary condition, Dr. Taylor stated that very careful drainage is necessary to get rid of anopheles larvæ, as the smallest collection of water left will harbour them. During the attempt to get rid of anopheles from Freetown, the smaller pools are merely swept out, but the larger pools are treated with some culicicide, either crude kerosine or crude creasote oil. By this means the larvæ are killed off when quite small. Formalin, corrosive sublimate, copper sulphate, carbolic acid, &c., did not prove so efficient larvæ destroyers as kerosine or creasote oil. A little oil is simply poured in a pool and the water well stirred up, so that a film of oil spreads over the whole surface. Crude kerosine thus

applied will last a week, if no more rain comes. A half drachm of crude creasote oil, if added to a pint of water in which there are adult larvæ, and the water stirred up, the larvæ will die within five minutes. Pupæ are more resistant and will live for an hour or more, especially if they are on the point of hatching. Should, however, they succeed in hatching, the adult insects cannot fly away, for they are caught in the film on the surface the moment they are free.

EDWARD HENDERSON, M.D.Edin., F.R.C.S.E. (late of Shanghai, China).

Heat Apoplexy.

Injurious effects from exposure to a high atmospheric temperature are divided into (a) those in which exhaustion with a normal or lowered temperature and little or no loss of consciousness prevails, and (b) those in which a high temperature and more or less complete loss of consciousness are the distinguishing features. The latter class is described under the names of thermic fever, siriasis, heat apoplexy, &c., and Dr. Henderson preferred to discuss it under the name of "heat apoplexy."

The cases seen by Dr. Henderson in Shanghai occurred amongst the foreign population, and chiefly among those visiting the port. Amongst the native Chinese heat apoplexy is a disease of comparatively rare occurrence, and he had never seen a case amongst foreign women and children.

Heat apoplexy develops more frequently in the house than in the open, and it seems to develop, or at least reach its climax, during the night. Alcohol was a complication in the greater number of the cases of heat apoplexy. The mortality was considerably above the 50 per cent. spoken of in text-books. The patients were all adults; the temperature was 107° or over, and the greater number were alcoholic. Dr. Henderson advocated the early and efficient application of external cold, and he showed photographs of, and described exactly, a douching couch he had employed with great benefit. Heart failure is the common cause of death in heat apoplexy, and it is necessary to guard against reducing the body temperature below 102° or 103° whilst applying cold.

ANDREW DUNCAN, M.D., F.R.C.S., M.R.C.P. (Physician to the Seamen's Hospital Society, London).

The Prophylaxis of Sunstroke.

From personal experience of sunstroke in India, Dr. Duncan gave details of a plan of treatment which answered admirably.

During several succeeding years he suffered from severe headaches, and during four hot seasons he had in addition intolerance of light and a tendency to unconsciousness. It was suggested to Dr. Duncan that the actinic rays of the sun and not the heat rays were the active agents in producing sunstroke, and that were the body enveloped as a photographer treats his plates, using always an orange-yellow wrapper, that the effect of the actinic rays on the body would be counteracted. Dr. Duncan, acting on this suggestion, wore an orange-yellow shirt, placed a similar coloured lining inside his service helmet and

inside the coat over the spine. After the use of this coloured material Dr. Duncan never again felt the influence of the sun to be overpowering.

(To be continued.)

The British Guiana Medical Annual for 1902.

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[The contents of this valuable annual we should wish to reprint in full. As, however, space is limited, we must content ourselves with placing before our readers in this issue a few extracts, and hope in subsequent issues to notice the remaining papers at some length.—Ed. J.T.M.]

FILARIAL LYMPHANGITIS.

By GEORGE C. LOW, M.A., M.B., C.M.(Edin.).

Cragg's Scholar, London School of Tropical Medicine.

CLINICAL FEATURES.

THE onset of an attack of filarial lymphangitis may be provoked, especially in the old-standing cases of filarial diseases, by any trivial scratch or abrasion, getting wet or bathing in the sea, and is usually sudden. In some cases the individual may go to bed quite well at night, and suddenly wake up feeling

chilly or even with a severe rigor upon him. Along with this sudden rise of temperature, which may reach 105° or 106° F., severe headache, sickness and vomiting, and often very marked delirium appears, the vomiting and general discomfort being more marked in the lymphangitis associated with filarial orchitis. If the case has never suffered from an attack before, and has no definite evidence of filarial disease, symptoms of pain localised in a limb, testicle or other part, or the formation of an abscess, at once puts one on one's guard, and leads one to make a careful local examination. The commonest feature complained of is pain in the glands of the part affected, and when the leg is this part, it is seen that the femoral group is tender, enlarged, and very painful on pressure. At the same time the limb is noticed to be swollen, generally only in the lower part, and a dusky redness is seen spreading upwards along the line of the lymphatics, these vessels feeling hard and cord-like to the touch.

After one or two days of high fever the temperature falls rapidly, and the patient is comparatively well; the delirium and headache have gone, the lymphangitis gradually becomes less, and the swelling and tension in the course of the next few days in many cases disappears completely, though in others a certain amount of thickening is left, which, when added to in subsequent attacks, eventually passes into the early stage of elephantiasis. When an abscess forms and complicates the case the temperature may not fall, or if it does, rises rapidly again and keeps high until the cavity is opened and drained; after this recovery quickly takes place.

DIAGNOSIS.

Practically speaking the only disease that can be mistaken for filarial lymphangitis in the tropics is malarial fever, but the local features of the former, apart from a blood examination, are so characteristic that mistakes should not arise. As there is no indigenous malaria in Barbados the diagnosis, as far as that island is concerned, becomes more simple still, and as the three cases were all natives who had never been out of the island, no possible doubt could arise as to the identity of the disease.

As will have been noticed already, the blood examination of such cases may give a negative result as regards filarial embryos, and that on several different occasions, but even without this help the local manifestations are quite sufficient.

In malarial cases the only point of resemblance is the rigor and sudden rise of temperature, but the examination of the blood on the admission of the case should show the plasmodium of that disease, and the temperature chart will be quite different in nature, having a daily rise for quotidian fevers and a rise on the third day in tertian fevers; further, there will be splenic tenderness, possibly enlargement, congestion of the liver, and an entire absence of lymphangitis and swelling of glands. It is possible that a filarial case might have malaria as well; but then the double set of symptoms would be present, the plasmodium would be found in the blood, and careful observations of the progress and the result of treatment would still further separate the two from each other.

PATHOLOGY.

The pathology of those attacks of lymphangitis is not yet thoroughly worked out, what part the adult the adult worms play in them being still uncertain. It is the exception to find embryos circulating in the blood, and that even in cases undergoing their first attack, such as occurred in several cases which I have examined. It may be that the death of the parent worms acts as the exciting cause for the attack, setting up some septic irritation which affects the lymphatics of the part, or at least if the adults have not died, the passage for the embryos into the blood must be completely shut off. Whether or not it then requires a fresh infection before another attack can take place can only be discovered by series of detailed *post-mortem* examinations of such cases, but as they never practically die from an attack of filarial lymphangitis it will be very difficult to determine this. In other cases embryos are sometimes found in the blood, but then those may be coming from adults lying in the lymphatics far removed from the seat affected.

Whatever the cause the lymphangitis would seem to arise first, either spreading downwards or in other cases upwards from the extremities, the fever, high temperature, and vomiting being results of this.

A certain class of cases in Barbados is known "as fever and ague" of the abdomen; those are rare and very fatal, but as far as I can ascertain no *post-mortem* examinations were made, and the conditions of the patients as to whether they were suffering from definite filarial diseases, such as orchitis, lymph scrotum, or varicose groin glands were not stated. It is conceivable they might have been caused by lymphangitis of the large vessels in the abdomen, complicated possibly by abscess formation; but until *post-mortem* examinations are made this is only theorising.

The lymphangitis tends to subside rapidly, even without any treatment, and, unless an abscess forms, quickly resolves and disappears, sometimes permanent thickening being left and sometimes not.

TREATMENT.

This is simple. If the bowels are constipated they should be freely opened by some purgative. The patient must be kept in bed, put on milk diet, and the limb should be elevated slightly, and some antiseptic lotion such as 1 in 40 carbolic, or 1 in 2,000 mercuric chloride applied. In the orchitis cases the scrotum should be put in a suspensory bandage and also have an antiseptic dressing applied. If abscess appears it should be opened by the usual surgical methods at once, and completely drained. In the cases where the swelling tends to remain and become permanent, massage may be employed, and an elastic bandage used. Drugs are not of much use except to alleviate symptoms. Quinine and antipyretics have been tried; the former does not influence the fever, and the latter, though relieving the headache and discomfort, do not really benefit the condition. If the discomfort is extreme they should be given a trial, and if the pain is very severe, opium may be given.

ERRATUM.—In Dr. H. ZIEMANN's article on *Lemadera*, in our issue for August 1, read *Hæmoglobinuria* for *Hæmaturia*.

FEVER CASES.

By C. P. KENNARD, M.D.(Edin.), M.R.C.S.(Eng.).
Government Medical Officer.

As there is no doubt that many fever cases are not malarial, and in order to find out the fluctuations in the malarial cases during the thirteen months from August, 1900, to August, 1901, I examined all cases complaining of fever, or who had fever, admitted to Pln. Marionville Hospital, Wakenaam, by testing their blood, and taking their temperature charts and cases; these latter were used again for reference if they returned.

The forms of malarial parasites found, correspond to those described by Dr. Manson as benign quartan, benign tertian, and malignant tertian (æstivo-autumnal). I have not found what he describes as the malignant unpigmented.

In some cases of fever I found, small actively moving bodies mostly in the shape of small rod- or pear-shape bodies, and frequently in couples in the red blood corpuscles; as these bodies never show pigment or would not stain by the ordinary malarial stains, and as quinine had not much action on the fever or bodies, I did not class these as malarial. I have, however, found these bodies in a few cases showing little or no fever, so I am doubtful at present if they may be pathological.

FEBRICULA.

Numerous cases of fever occur where there is some shivering, followed by hot fever lasting some hours, and maybe some sweating after this. The case is given a dose of mist. mag., and there is no recurrence. No malarial parasite being found, no quinine was given. These were classed as febricula; this class of fever corresponds to many cases seen in every district.

LOW FEVER.

Many cases of "low or inward fever" were tested both from the hospital and outside, and it is very exceptional to find the parasite in these cases; as most of us have had it at some time or other the symptoms of chill, headache, aching of the limbs, lassitude, with some feeling of heat, are well known. It may be from hot oppressive weather, often dyspepsia and torpid liver, often from worry, but it is very exceptional to be from malaria. There are numerous other causes for febricula, in fact anything that may be unusual; worms in children and adults, and dysmenorrhœa in women are frequent causes.

I am inclined to think there is more quartan fever about than is supposed; it is just the fever that a man may have a distinct attack, lie down till it is over, and then go about his business without feeling particularly bad. Quinine does not appear to have such a rapid action on this parasite as the pernicious form. The spleen was enlarged in eight of the cases; in none was there any pain or tenderness. In no case was anæmia marked after it.

BENIGN TERTIAN FEVER.

There were 23 cases of benign tertian fever, four having recurrences, 8 men, 5 women, and 10 children. In these cases the fever is usually more pronounced than the benign quartan; it rarely keeps above the

normal continuously for more than two days; it usually shows a sharp rise and a fall to normal in a few hours, returning the next day or the day after; it is frequently irregular as to time and height of temperature, a regular every other day rise is unusual; sometimes very little rise is seen.

PERNICIOUS OR MALIGNANT MALARIAL FEVER.

There were 81 cases showing the form of parasite corresponding to that described by Dr. Manson as malignant tertian or æstivo-autumnal; 53 men, 16 women, 12 children: ten of these were recurrent cases, one four times, one three times, and the rest twice at not long intervals. The fever shown was very various; in a few there was very little fever, in others it was high and continuous for two to five days under treatment; some had a daily sharp rise with ague and sweating, others had an every other day similar rise; some had no ague, in others there was little sweating, and in two cases there was marked ague and sweating at the same moment with a high temperature.

This fever undoubtedly causes more constitutional disturbance, and the patients are more "knocked over" afterwards, than in the benign forms. Some patients who had it mildly, however, appeared little affected; cases that had the benign form severely also had the pernicious form severely. Vomiting bile and "biliousness" was very common. Severe headache, which may not depend on the height of temperature, and pains in the limbs were present in nearly every case. Pain in the splenic region is rarely complained of and tenderness there rarer. Severity of the attack does not seem to depend on the abundance of the parasite seen in the blood, or the condition of the spleen, or whether previously anæmic, but more on the "biliousness" of the subject. An advanced case of phthisis had a rather sharp attack, and made a perfect recovery; a case of chronic Bright's disease was not specially affected by it: he was not given quinine; the albumen had diminished when he left hospital, and no crescents developed.

THE MOSQUITO AND MALARIA.

A LECTURE DELIVERED BEFORE HIS EXCELLENCY THE GOVERNOR, AND THE ROYAL AGRICULTURAL AND COMMERCIAL SOCIETY OF BRITISH GUIANA, FEBRUARY 13TH, 1902.

By A. T. OZZARD, M.R.C.S.(Eng.), L.S.A.

[We reprint the paragraph on the "Parasitology of Malaria" as a model for others to go by when explaining this intricate subject to laymen—a most important duty.—Ed. J.T.M.]

PARASITOLOGY OF MALARIA.

The malarial parasite belongs to the large group "Protozoa." It possesses an extremely complicated and interesting life-history, the working out of which has perhaps stimulated more thought than that of any other pathogenic micro-organism.

It is parasitic in man and in a certain genus of mosquito (*Anopheles*); the former is its intermediate host and the latter its definitive host. In its human

host it undergoes various stages of development within the red corpuscle. Here during its stage of growth it is a tiny unicellular organism (1-8 micromillimetres diameter). Like other protozoa, it is made up of cell protoplasm, nucleus and nucleolus. It moves, it grows, it absorbs and assimilates food material, and it reproduces itself. Whilst within the red corpuscle the parasite exhibits amoeboid movement, which enables it to change its shape and also its position in the red cell. The parasite assimilates the hæmoglobin of the red cell in which it is contained and elaborates from it characteristic melanin granules, the so-called malarial pigment.

The by far most interesting feature in the life-history of the malarial parasite is its method of reproduction. It possesses, like the coccidiæ, and some other forms of animal life, two modes of reproduction, one endogenous and the other exogenous. The former is by sporeformation, and is an asexual process. This process can be carried on and obtain maturity in man without the intervention of the mosquito. The latter is a sexual process which can only be completed by passage through a certain genus of mosquito. The asexual cycle was first carefully described by Golgi, and has been termed the Cycle of Golgi. The sexual cycle is due in the main to the brilliant researches of Major Ronald Ross, late of the Indian Medical Service, and has been called the Cycle of Ross.

The necessity for the Cycle of Ross for the perpetuation of the parasite is evident, because in the absence of an intermediary carrier, such as the mosquito, the malarial parasite would be unable to pass from man to man, and on the death of its human host would die also.

The diagram illustrates in a schematic way the phases of development of the endogenous and exogenous life cycles of the malaria parasite respectively.

If the blood of a patient be examined during an attack of malarial fever, say, of the benign tertian type, and if this examination be made shortly after a rigor, a certain number of the red corpuscles will be found to contain tiny amoeboid bodies. They are pale and indistinct. They are constantly changing shape, protruding and retracting pseudopodia. At this early stage of development there will be no pigment granules present. If the blood of the patient be examined again after an interval of twelve hours the parasites will be found to have increased considerably in size and to occupy now about one-quarter of the red corpuscle. They are still actively amoeboid. A few granules of pigment will now be observed in the protoplasm of the parasite. If the blood examination be repeated after a second interval of twelve hours the parasites will be seen to have increased still further in size and to contain more pigment. Amoeboid movement will now be becoming less active. If these blood examinations be continued at serial intervals a gradual increase in the size of the parasite can be traced, until a short time before the next rigor they come to occupy nearly the whole of the red corpuscle. At this stage the pigment will be scattered throughout the protoplasm of the parasite, and it will be abundant and coarse in grain owing to the agglomeration of separate granules. If an examination of the blood be made at the outset of rigor, the parasites will be found to be at the sporulating stage showing the so-called rosette bodies. The shape, size, number,

and arrangement of these spores vary with the species of the parasite. In the benign tertian infection, of which we are now speaking, the sporulating body is made up of 15 to 23, in the quartan 6 to 14, and the malignant 5 to 12 spores. The final stage, which is best observed in quartan infections, is the rupture of the red cell and the diffusion of the spores in the plasma. The corpuscular debris and pigment granules are rapidly taken up by phagocytes, whilst the free spores rapidly gain entrance to fresh red corpuscles and become the new generation of young endo-corpuscular parasites. The young intracorpuscular bodies are termed *amæbule*, the rosette bodies *sporocytes*.

In addition to the bodies just described there will be found in the blood in the case of the benign fevers large pigmented spheres, and in the case of the malignant fevers the well-known crescent bodies. These represent the form of the parasite which require to enter the stomach of the mosquito (*Anopheles*) in order to attain full development.

In the case of the benign fevers the spheres make their appearance in the peripheral circulation within one or two days from the onset of fever, usually during the apyretical period. The crescent bodies, however, do not as a rule appear in the blood until about one week from the onset of the fever, sometimes later than this.

If the blood of a patient, known to contain crescents, be watched, these bodies will be observed to become gradually kidney-shaped and oval, and then spherical. A certain number of these newly-formed spheres will be seen to become agitated, and the pigment they contain will become extremely active and dancing. Suddenly one or more flagella are shot out. These filaments possess a rapid vibratory and lashing movement, and at first, on account of their great activity, they can be seen with difficulty. Gradually movement becomes less active. The flagella will then be seen to be about four or five times the length of the diameter of the red corpuscles, colourless, free from pigment, and bulbous or beaded in some point, or throughout their continuity. A certain number of spheres, however, will remain quiescent and will be observed not to flagellate. These latter are the females (macrogametocytes-vessels containing big wives), the former the males (microgametocytes-vessels containing little husbands). The difference in the sex of the crescents can be readily distinguished by a certain method of staining the blood. According to Ray Lankester, the flagella must be regarded as homologues of the spermatazoons of higher animals. The next stage, the flagella break away and approach the non-flagellating spheres, which at one point have developed a small papilla. At this point a flagellum enters, having previously become rigid and motionless. After its entry a certain amount of disturbance takes place in the sphere. It soon, however, becomes quiescent and then elongates, the pigment becoming gathered at the posterior or thicker extremity of the now fertilised cell. It now assumes the shape of a spear head and becomes actively motile, moving freely in all directions (travelling vermicule). It is believed that it is this body that pierces the stomach of the mosquito and develops into a zygote (zuywtos-yoked or wedded).

The next stage can be readily observed in the case of human malaria. If a number of Anopheles be made to bite an individual whose blood contains crescent bodies, and if these mosquitos be killed at intervals and examined, the following changes may be seen: If one be killed a few minutes after feeding, the blood contained in its middle intestine will be found to contain flagellating and non-flagellating spheres; if a mosquito be killed on the second day after haustellation and the wall of its middle intestine examined pigmented spheres (zygotes), about 7 or 8 micromillimetres in diameter, will be seen lying in and disassociating the muscular fibres; if infected mosquitoes be examined on subsequent and successive days further changes in the development of the parasite can be traced out. The pigmented spheres increase rapidly in size and become surrounded by a capsule. They will be seen to contain smaller spheres (zygotomeres) which apparently divide and sub-divide, and each of which contains chromatin matter which also divides and sub-divides. Eventually bud-like processes develop on the surface of these little spheres giving them a hedge-hog appearance; usually 10 or 12 such spheres (blastophores-germ-carrier) are formed. These bud-like processes gradually become elongated and sickle-shaped. They finally fill the cell completely, the remains of the secondary spheres disappearing. As these large spheres mature they protrude through the outer surface of the wall of the middle intestine as warty processes, and in heavy infection they are present in large numbers. (Their size varies from 40 to 60 micromillimetres). Eventually they rupture on their outer surface, and the sickle-shaped bodies (sporozoites) they contain become free and are carried in the body fluid of the mosquito to its salivary glands, where they appear to be filtered out by the middle lobes of the glands. It is these bodies which are the actual source of infection to man, and they have been traced as far as the end of the proboscis of the mosquito.

The mosquito cycle occupies a time varying between six and sixteen days or even longer, depending on temperature and possibly on other factors.

DESCRIPTION OF A FEMALE PARENTAL FORM OF THE FILARIA DEMARQUAYI.

By A. T. OZZARD, M.R.C.S.(Eng.), L.S.A.

THE following description unfortunately fails to definitely settle certain important points, owing to the faulty condition of the specimen when received. It was found impossible to sufficiently clarify and stain it so as to bring out certain important characteristics, notably of the tail-end.

A female parental form of *F. Demarquayi* was some-time back sent to me by Dr. Otho Galgey, of St. Lucia. The specimen was a dead dried one on a glass slide, and not in spirit. I endeavoured to clarify it by means of absolute alcohol and oil of cloves; but not so successfully as could have been wished. It was then stained with logwood.

The specimen is a complete one, and measures, in its dried condition, about $2\frac{1}{4}$ inches. At its broadest point it measures about $\frac{1}{100}$ inch.

The head and tail-ends are deeply stained, interfering somewhat with a detailed examination.

The head is rounded, and an indication of a minute mouth can be made out with a high power. No differentiation of alimentary canal into oesophagus and intestine can be made out. To all appearance the head is similar to that of the female parental form of *Filaria perstans*, as described by Dr. Daniels in the *British Medical Journal* of April 16th, 1898.

At a distance of about $\frac{1}{10}$ inch from the head, the specimen is unfortunately broken; it is possible that at this point the vagina opens externally, as at no other part can any indication of the termination of the vagina or ovarian tubules be made out.

An alimentary canal is clearly seen running the whole length of the specimen, and to all appearances this canal terminates at the extreme tip of the tail, instead of in a papilla, such as is the case in the parental forms of *F. nocturna*, *F. perstans*, and the so-called *F. Ozzardi*. It is true that at a distance of about $\frac{1}{100}$ inch from the tip of the tail there is a slight divergence in the outline of the animal; but under high powers even, no appearance of the intestine terminating there is suggested, nor does it appear to be anything more than a slight irregularity in outline.

The absence of an anal papilla, therefore, is in marked contrast to what obtains in the parental forms of *F. nocturna*, *F. perstans*, and the so-called *F. Ozzardi*.

The tail is sharply curved for the last $\frac{1}{100}$ inch or more, similar to the curves of the tails of the parental forms of other filariæ.

The tip of the tail appeared to be made up of four papillary processes (two seeming to overlap the other two) instead of two processes, as described by Dr. Daniels in the female parental form of *F. perstans*, or the bulbous termination of the female parental form of *F. Ozzardi*.

As in other parental forms, there are two ovarian tubes containing ova and embryos in various stages of development. The embryos are so coiled up and crowded together that it is almost impossible to say whether their tails are sharp or blunt; but as Dr. Galgey ascertained that the host during life-time contained numerous *F. Demarquayi* and no blunt-tailed embryos, it is not of much importance.

The breadth of the ova varied from $\frac{1}{500}$ to $\frac{1}{100}$ inch. The breadth of the ova of *F. Bancrofti* as given by Lewis, varies from $\frac{1}{1500}$ to $\frac{1}{100}$ inch. In other words, the ova of the parental form of *F. Demarquayi* are about equal in breadth to that of the embryo *F. nocturna* itself. So that it is easily understood that diseases such as elephantiasis do not occur in those the hosts of *F. Demarquayi* or the other minute filariæ, such as *F. perstans*; whereas they may do so in those the hosts of *F. nocturna*, in consequence of the greater breadth of the ova of the latter.

It is unfortunate that the tail-end of this specimen could not be clearly defined. Dr. Daniels in a private letter to me, states that in a similar specimen (female parental form of *F. Demarquayi*) examined by him, the tail ended in two rounded knobs, quite different to what obtains in the parental forms of *F. Ozzardi*.

It is of the utmost importance, therefore, that clear descriptions should be given of the parental forms

(male and female) of both *F. Demarquayi* and the so-called *F. Ozzardi*.

Dr. Low, of the London School of Tropical Medicine, during his recent visit to the West Indies, established the fact that the embryo forms of *F. Demarquayi* and *F. Ozzardi* are absolutely identical; a point about which there was previously considerable doubt.

The fact that the embryos are similar in size and appearance, however, is not sufficient to establish their identity as a species. For as in the case of *F. nocturna* and *F. diurna*, although the embryos are absolutely identical in appearance, owing to their difference in periodicity they are classed as two distinct species. If, therefore, it can be established that the parental forms of *F. Demarquayi* and *F. Ozzardi* present certain different well-marked characteristics, or, on the other hand, are absolutely similar in all respects, their identity or otherwise will at once be settled.

It is worth noting that the distribution of *F. Demarquayi* and *F. Ozzardi* is entirely different. Whereas in British Guiana the latter embryo is only found at distances up the big rivers and not along the coast-line, in St. Lucia and St. Vincent there is no such characteristic distribution of the *F. Demarquayi*.

Current Literature.

PLAGUE.

PREVALENCE OF THE DISEASE.

INDIA.—During the weeks ending July 12th and 19th, the deaths from plague in India numbered 1,158 and 1,100 respectively. In the Punjab the number of deaths from plague had during these weeks fallen below 100. In the Bombay Residency there is evidence of a recrudescence of the disease.

EGYPT.—During the weeks ending July 27th and August 3rd, the fresh cases of plague in Egypt numbered 10 and 5, and the deaths from the disease 5 and 2 respectively. In Alexandria most of the cases of plague occurred, but from Toukh, and Damanhour, cases are also reported.

CAPE OF GOOD HOPE.—During the weeks ending July 12th and 19th, the number of fresh cases of plague in Port Elizabeth numbered 3 and 0, and the deaths from the disease 2 and 1 respectively.

HONG KONG.—During the weeks ending July 26th, August 2nd and 9th, the fresh cases of plague numbered 33, 31 and 14, and the deaths from the disease 29, 34 and 13 respectively.

MAURITIUS.—During the weeks ending July 31st and August 9th, the fresh cases of plague numbered 6 and 2, and the deaths from the disease 5 and 1 respectively.

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Archives Russes de Pathologie, de Médec. Clinique et de Bacteriologie.
Australasian Medical Gazette.
Boletín de Medicina Naval.
Boston Medical and Surgical Journal.

Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Brooklyn Medical Journal.
Caducée.
Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Esercito.
Hong Kong Telegraph.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
Journal of the American Medical Association.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal
Medical Brief.
Medical Missionary Journal.
Medical Record.
Medical Review.
Merck's Archives.
New York Medical Journal.
New York Post-Graduate.
Pacific Medical Journal.
Polyclinic.
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The Journal of Tropical Medicine.

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Original Communications.

SOME CLINICAL NOTES ON A EUROPEAN PATIENT IN WHOSE BLOOD A TRYPANOSOMA WAS OBSERVED.

By R. M. FORDE, L.R.C.S., &c.
Colonial Surgeon, Gambia, W. Africa.

H. K., aged 42, European, Master of Government steamer, River Gambia, West Africa. Six years in the service of the Gambia Colony.

Previous health in West Africa: With the exception of one or two ordinary attacks of mild remittent fever he always enjoyed good health on the Coast up to the onset of the present illness, and was what I should describe as a man of robust constitution, living a most regular and steady life.

Mr. K. reported himself sick on May 10th, 1901, stating that he was feeling feverish and out of sorts for some days past. I admitted him into the Colonial Hospital, Bathurst, when I found him suffering from what I at first took to be an ordinary attack of malarial fever and put him on small doses of quinine accordingly. After a few days' treatment I noticed very little change in the course of the temperature, and took some slides of blood for microscopical examination, in which I found no malarial parasites, but found in nearly every specimen, and on frequent subsequent examinations, small worm-like, extremely active bodies, which I prematurely pronounced a species of filaria; this conclusion, however, became doubtful after repeated observations of the parasite, and at the same time I could not resist associating the peculiar course of the illness, its symptoms and resistance to treatment, to the presence of these worms, whatever they might be. At the end of three weeks the patient was invalided to Europe, and I strongly advised him to present himself for treatment at the Liverpool School of Tropical Medicine (his home being in Liver-

pool) and to write and let me know when he had done so, but unfortunately I never heard any more about him until his return from sick leave in December. In the meantime, Dr. J. Everett Dutton, from the Liverpool School of Tropical Medicine, arrived at Bathurst, and having told him about the case and what I had seen, he examined the patient's blood on the first opportunity and discovered the same parasite I had found some months previously, which he at once recognised as a species of Trypanosoma.

Symptoms.—As seen by the accompanying charts the temperature is not that of malarial fever, it is irregularly intermittent with, as noted by Dr. Dutton, two or three days of normal, or subnormal, periods, it was not affected in any way by drugs, large doses of quinine and the usual antipyretics having only a temporary, if any, effect on its course.

The skin was as a rule dry, with later, irregular patches of a congested or cyanosed character appearing on different parts of the body, the colour slowly returning after pressure. An oedematous condition made its appearance early, most marked on the face below the eyes, producing a characteristic fulness in that region, which varied in degree from a scarcely noticeable swelling to well-marked puffiness; this condition was also noticed in the lower part of both legs and around the ankles, but only in a slight degree.

The respirations were always above the normal rate, ranging from 20 to 30 per minute, periodical accelerations recurring quite independently of any rise of temperature, and whilst the patient was quietly lying in bed.

The pulse was also accelerated, ranging from 70 to 120; there seemed to be no relation between the rate and the temperature, sometimes it was highest when the latter was subnormal, at others a maximum temperature was accompanied by a minimum pulse. The beat was always strong and regular. I could detect nothing abnormal in the position of the heart, but the sounds had a peculiar muffled character.

The urine was passed in fair quantity and was

would have a beneficial effect; he returned, however, looking no better, having been taken ill on the outward voyage with what was said to be an attack of pneumonia. After two or three weeks he felt sufficiently strong to return to duty. All this time the irregular temperature and the other symptoms, with a feeling of weakness in the lower extremities, persisted, but to a lesser degree.

Treatment.—The only drug that produced any good result was arsenic (Fowler's solution) in graduated doses, there was a general marked improvement under its influence, but all the symptoms returned after it was discontinued for some time. Rest and light nutritious diet were the chief means of sustaining the patient's strength.

Remarks.—The chief characteristics of this case were as follows: (1) The irregular intermittent temperature; (2) the oedematous condition of the face and lower extremities; (3) the rapid and variable pulse and respiration, unaccompanied by any evident cause; (4) loss of weight with marked debility, wasting and lassitude; (5) the persistence of these symptoms and their resistance to treatment.

I have lately noticed similar symptoms in natives, but have not succeeded in finding the parasite referred to in this case in the few cases examined; Dr. Dutton has, however, found the worm in the blood of a native child three years old, but who showed no symptoms of disease.

THE PARASITE.

The accompanying photograph, kindly sent to me by Dr. Dutton, shows the appearance and position of the parasite in a stained preparation, and he informs me that it presents all the characteristics of the genus *Trypanosoma*, and *morphologically* is very nearly related to *T. brucei*.

SOME POINTS IN CONNECTION WITH THE OPERATION FOR THE EXTRACTION OF CATARACT IN INDIA.

From an Experience of over 2,000 Cases.

By Capt. R. H. ELLIOT, M.B., B.S.Lond., F.R.C.S.Eng., &c., I.M.S., Madras.

PRELIMINARY CONSIDERATIONS.

IN selecting cases for cataract extraction one cannot afford to neglect the *condition of the conjunctiva*. Even the slight forms of conjunctival congestion so commonly met with in Indian practice require "watching"; if there is any morbid secretion on the lids in the early morning, preliminary treatment is necessary. Silver nitrate is most generally useful, but there are not a few cases which, while intolerant of stronger remedies, will quickly yield to mild lotions such as *lotio acidi borici*.

In a series of 500 cases, in which I was inclined to think lightly of slight departures from the normal in the state of the conjunctiva, thirteen out of 261 of such cases, or 4.98 per cent., showed septic infection with suppuration, against six out of the remaining 239, or 2.51 per cent. In a succeeding series of 250 cases, conjunctival complications figured at 172, in 100 of which treatment lasting from three to thirty-one days was given before operation; in two of these,

or in 2 per cent. of those submitted to preliminary treatment, suppuration manifested itself. In the remaining 150 there were three suppurative cases, or again 2 per cent. It would thus appear that attention to the conjunctiva had eliminated the extra risks consequent on an unhealthy state of the membrane.

It is seldom necessary to interfere with a *pterygium*, provided that the conjunctiva is otherwise healthy, and that one can avoid cutting the growth during one's section. I find that out of seventy-six cases of pterygium occurring in 827 cataract extractions, five gave trouble; in only one of these was convalescence seriously delayed; in this case the conjunctiva had required treatment beforehand, and was exposed to the strain of prolonged bandaging, owing to the patient bursting his section by rubbing the eye. In all five of the cases good vision was obtained, as also in the remainder of the seventy-six. In two instances the pterygia were transplanted some time before extraction. One is very loth to operate for cataract in cases of recent corneal mischief, even though the eye appears quite quiet again; a long interval should be allowed to elapse first. A dexterous operator will be able to so place his incision as to obtain an iridectomy in any required position, and will thus be able to deal with a central opacity and with the cataract at one sitting. It is very inadvisable for anyone to attempt this who has not absolute confidence in his powers; he will be better advised if he does an iridectomy in the most suitable direction as a preliminary operation, and deals with the cataract a month later.

Posterior synechia is always a serious complication of cataract. I find that I met with it nine times in my last 827 cases. In seven cases excellent results were obtained, although in one about one-sixth of the vitreous was lost during vectis delivery of the lens. In one case of complete posterior synechia, vision was raised from mere perception of light and darkness to ability to detect hand-movements at 3 metres. In one case vision was lost due to iritis. The loss of one eye in nine is justified by the results of the other eight. Only one case of anterior synechia appears among the same 827, though there was some iritis after extraction; the result was satisfactory.

In 2,000 cases I cannot remember any case in which *mischief in the lachrymal passages* has prejudiced the result of an operation for cataract; in the last 827 cases the complication occurred four times; in these, as in my previous cases, the duct was well dilated, and the parts brought into a healthy condition beforehand.

In suitable cases I would not hesitate to excise the sac as a preliminary measure, as is the custom of several of the leading continental surgeons I have met.

Considerable variation is met with in the *tension* of eyes requiring cataract extraction. In 827 cases ninety-nine showed an increase, and sixteen a diminution in tension; under this head are now being considered only cases which in other respects appeared suitable for operation, markedly pathological conditions are excluded.

The percentages of success work out at 89.9 in the eyes with raised tension and 81.25 in those with lowered tension, against 93.34 per cent. on the total of 827.

It is no uncommon thing in India to meet with *partially dislocated cataracts* which, in almost every case, are the fruits of the Mahomedan Coucher's work. If otherwise the case is favourable one should extract the lens. In the last five years I have operated on nine such cases, and with most encouraging results. It is well for the operator to remember that if the attempt is a failure he will almost certainly be credited with the whole responsibility. In such cases the principal danger is from loss of vitreous, the lens should be delivered in its capsule; this can usually be easily affected by manipulation after iridectomy, but should difficulty be encountered bold vectis delivery is indicated.

Not all cases of *irido-donesis* are due to dislocation; a certain percentage are met with in old shrunken lenses, and are presumably to be attributed to overstretching and relaxation of the zonule of Zinn; the treatment is as for partial dislocation. Of eight such cases, of which I have notes, seven recovered good vision.

There is a class of cases in which the *iris does not react freely to mydriatics*, sometimes dilating very slowly and partially, at other times dilating irregularly, and yet no evidence of iritis or of synechiæ exists; seven such cases, of which I have notes, made uninterrupted recoveries. It would be most inadvisable to attempt in these the simple operation.

Some years ago I freely tried the method of "*preliminary iridectomy*" as a separate operation, combined in many cases with massage to hasten maturation, but have ceased to employ these manoeuvres, as while dissatisfied as to their utility, I found them not free from objections. At the main operation one not infrequently finds that small posterior synechiæ, the result of the previous iridectomy, complicate delivery; then, again, there is the inconvenience to the patient, and the danger withal, of two operations instead of one. A distinguished Indian operator (now retired) used to make a downward and inward iridectomy in slow developing immature cataracts. I have seen old patients of his, who, thanks to this treatment, enjoyed many years of useful vision; but the coloboma in this position is a distinct disadvantage *after extraction*, and I consider the method vastly inferior to that of MacKeown, of Belfast, of which after trying it in a large number of cases, I can speak in the highest terms.

Diabetes and albuminuria have been urged by some as a bar to operation. I have operated with success in both of these conditions, and provided the general health of the patient is good, I do not consider either of them as forbidding extraction. The same may be said of old age. My oldest patient in the 2,000 cases was said to be about 90; she looked like a shrivelled old monkey, but she was full of vitality and made an excellent recovery, using her restored sight for the first time to steal a neighbour's necklace the night after operation. An active European lady of over 80 did equally well, and insisted on leaving the private hospital she was in to return home a fortnight after operation.

On the other hand, any conditions which depress the general health indicate a postponement of the operation till the patient is in better condition. This

is well illustrated by a recent case in which the patient, a weakly man, was doing well on the tenth day, and appeared to be out of danger, when a sudden violent attack of diarrhoea came on and the cornea suppurated before the patient's death, which took place from exhaustion.

METHOD OF RUPTURING THE CAPSULE.

I prefer the use of a Bowman's needle as the first stage of the operation to any other method; it has the following advantages:—

(a) It is a very easy method and enables the surgeon to see clearly what he is doing. The iris is therefore in no danger of being injured. With a very moderate amount of skill a needle can be introduced into even the shallowest chamber without catching in this membrane.

(b) The laceration of the capsule can be effected without any need of haste, since at this stage there is no risk of an escape of vitreous, even should the patient attempt to squeeze the eye. The operator can make as large a rent in the capsule as he wishes, and can place it with ease in the exact position he fancies. This balances the advantages claimed for the capsule-forceps of removing a portion of capsule, for with a free, well-placed aperture the edges retract widely during and after the escape of the lens.

(c) The surgeon can confirm his diagnosis as to the nature of the cataract, for the needle in an experienced hand is practically a probe, and thus its use throws valuable light on the after stages of the operation.

(d) There is no angle for the lodgment of dirt, as there is in the cystitome and capsule forceps, and therefore sterilisation is more easily secured.

There are a few points of interest as to the method of needling, and as to the information which this proceeding affords. It is essential that the needle should be *very sharp*, and that it should have cutting edges.

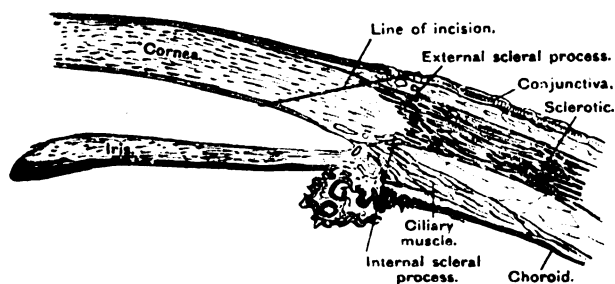
Into the cortico-nuclear cataract the needle edge sinks easily. As a rule, the capsule of a Morgagnian cataract readily bursts when incised, giving exit to a cloud of turbid fluid, which, though it may obscure the details of the anterior chamber, compensates for this disadvantage by very sensibly deepening the chamber, and thus enabling the operator to avoid cutting the iris. In a few cases one finds the Morgagnian capsule extremely tough. If so the needle must be withdrawn and the membrane lacerated with the point of the knife before the section is made. Such cases are, however, fortunately rare.

The feel of a hard cataract is characteristic, and in the endeavours to lacerate its capsule the lens may be seen to recede towards the posterior chamber. In such cases the greatest care must be exercised, so as not to apply undue force; the point of the needles should be introduced obliquely into the cataract, and the laceration effected by cutting forward with the edge of the needle into the anterior chamber. Should this precaution be neglected, and undue backward pressure made, the ligament of the lens may easily be ruptured, in which case a vitreous escape will probably occur.¹

¹ I have ventured to quote freely here from my paper published in the *Indian Medical Gazette* in July, 1897.

THE SECTION.

One can only explain the large number of varieties of section employed by different operators on the assumption that the detail is not a very important one. My own preference is for a section in which the knife enters, emerges and cuts out along the line of junction of the external scleral process with the cornea. A glance at the diagram will show that the section impinges on the sclerotic only along this line, passing through the cornea alone in the rest of its extent; at the same time a slight rotation of the axis of the blade backwards before emergence enables one to pick up a conjunctival flap in any desired case; such an addition is advisable in the case of old people, or in those whose feeble nutrition suggests a fear of delay in the healing of the section.



Modified from Norris and Oliver's system.

There is one point, however, of undoubted importance, viz., that the section *must be large enough*. During the last few years up-country medical men have from time to time written to ask my opinion as to the cause of failures they have met with in cataract extraction, sending me notes of their cases. In a large percentage of these there has not been the slightest difficulty in saying that the eye has been lost owing to the section being too small; their subsequent experience has proved the criticism to have been well founded. There are *objections* to too large a section, but there are *grave dangers* in one which is too small. An intelligent use of the needle whilst tearing the capsule will indicate the size of section required. In large, hard, bulky cataracts I do not hesitate to use a section just under half the circumference of the cornea in extent, and I have never seen any serious results therefrom.

We come next to the much disputed question of the advisability of choosing the "simple" or the "combined" operation. This is a question of much importance, and in a recent tour through Europe I have been at considerable pains to ascertain the views of leading surgeons on it, and to watch their own practice in this respect. Speaking broadly, the exponents of the simple operation hold that it is possible to select the cases in which iris prolapse is unlikely; their indications vary, but may be summed up thus: Given a patient in good health, not broken by age, free from any cause of straining, with a healthy eye, whose chamber is deep and whose pupil is active; given, too, good nursing, whereby the patient may be "immobilised" (in other words, saved all need of active muscular effort) for twenty-four to forty-eight hours;

given such conditions, and you may safely dispense with iridectomy.

There are very few advocates of the "simple operation" who, speaking from a large experience, will deny that they meet with a non-negligible percentage of prolapse, and with a farther quota of "displaced pupil." I do not deny that small series may be insufficient to show this factor. I have it on the best authority that Prof. Schnabl, of Vienna, has several times gone back to the "simple method," attracted by its advantages, and has as often abandoned it, owing to the occurrence of prolapse after a run of encouraging cases in which he had successfully escaped that complication. My own experience in the first 1,200 or more cases I did was very similar. A long run of cases in which no prolapse had occurred would be rudely broken by a painful percentage of prolapse. Several well-known surgeons have told me that their experiences has coincided with my own in this respect.

There is an important point which calls for notice before we proceed further. It is that the European surgeon works under conditions which we in India do not enjoy: I think that I might safely class most "tropical" eye surgeons with ourselves. We labour under the disadvantage that we *cannot* possibly effectually immobilise our patients after operation. To begin with, we have not the highly-trained nursing staff requisite for the purpose, and even if we had, the *native* would not submit kindly to the irksome control imposed. I am not speaking only of the coolie and the pariah, but I include the high caste and intelligent educated native, and I speak after having operated on a large number of the latter, both privately and in hospital. One meets with an occasional exception, but such do not affect the rule. Be it remembered, too, that the operation for cataract is a painless one, that the patient is otherwise in good health, and that even in Europe and with the best of nursing, the patient requires constant watching to save him from the consequences of his own indiscretion.

To appreciate the importance of the above remarks, we must first consider the causes of iris prolapse after cataract extraction. These may be divided into (1) predisposing, and (2) exciting.

Under *predisposing* causes I would class any factor leading to an impairment of the active contractility of the iris. Such a lesion may be present and recognisable before operation, or may be produced by over-stretching or tearing of the membrane during operation.

Under *exciting* causes we may class sudden movements of the patient after operation, and all causes which lead to straining. Such are the *very* conditions which good nursing controls for us, and which in India we find it so hard to guard against.

It is generally accepted that an iridectomy prevents prolapse of the iris by providing a safety-slucce through which the aqueous can escape when the section ruptures. If this were the whole of the explanation, I venture to think that the Indian patients who sit out in the compound discussing the details of the morning's operation with their fellows would suffer from prolapse much more frequently

than they actually do. There is undoubtedly another element in the case, viz., the "tone" of the iris. If this membrane, by virtue of its unimpaired activity, lies back in its usual position against the lens, the posterior chamber is reduced to its usual small proportions, and there is, accordingly, comparatively little "aqueous" dammed up behind it; hence it is less likely to be carried outwards by the rush of fluid when the chamber bursts; furthermore, its own muscular tone tends to keep it in its usual place, and does not allow every escaping stream to carry it up like a bellying sail.

When, on the other hand, the iris tone is lost, each outward rush of fluid *dammed up in unusual quantity behind it* acts on it at an advantage, by virtue of its position, and tends to carry the membrane before it, and so cause prolapse.

Many surgeons, recognising the risk of damage to the iris during operation, recommend a careful inspection of the pupil after delivery of the lens, with a view to the performance of iridectomy *then*, if the pupil shows any tendency to be displaced upwards.

Apart from the fact that iridectomy is hazardous after the lens is "out," I have not found this means of diagnosis satisfactory, since a perfectly round pupil, under the quiescent conditions prevailing in the empty chamber just after the operation, is *not* a guarantee against prolapse under the stress of a sudden rupture of the section when the chamber has had time to fill.

A valuable side-light on this lack of iris-tone can be obtained by considering a class of cases familiar to many cataract operators. I refer to those cases in which prolapse of the iris, not present at first, becomes slowly established during convalescence. A careful examination will reveal that these are cases in which a want of firmness in the section permits of a steady though imperceptible outflow in its direction; the iris, wanting in tone, becomes gradually drawn up into the wound and impacted therein.

Whilst many surgeons with European experience are at variance as to the advisability of omitting the iridectomy, I have not met *one* who would advocate the performance of the simple operation under the conditions we meet with in India. We *cannot immobilise* our subjects, and our risks are consequently *disproportionately* great. Such considerations have led me to adopt the combined operation as my routine in the last 800 cases I have operated on for the removal of the lens, and with very satisfactory results. In a series of 500 consecutive operations for primary cataract, I find that in 484 an iridectomy was performed, and in twenty-one of these there was some measure of iris prolapse, i.e., in 4.33 per cent. Of these nineteen recovered with good and two with poor vision. It is further noted that the symptoms of prolapse were not so severe as those met with in that complication after the simple operation. Again, in a later series of 250 consecutive cases of extraction for primary cataract, in which great attention was paid to the thorough replacement of the iris edges by means of MacKeown's irrigator, only six showed any impaction of the iris margins, and in only two of these was it necessary to remove the impacted edges and replace the iris. The percentages here work out at 2.4 per cent. for

the prolapses, and 0.8 per cent. for subsequent interference. Great care was exercised in both these series to avoid overlooking any impaction of the iris, however slight.

As to the objections commonly urged against the use of an iridectomy in cataract extraction, I cannot find, after carefully comparing the results of the two operations in opposite eyes of the same subject, that the average of visual result is better after the simple than after the combined operation; nor can I concede that mobility of the pupil is lost after the combined operation, for such is not the case: probably it is somewhat impaired, and this impairment may lead to some loss of post-operative accommodation, but the last-named condition is so seldom met with that this hypothetical loss need not much concern us. As to discomfort from the coloboma admitting an excess of light, I have seldom met with complaints from this defect, since I endeavour to make my iris-section narrow; the coloboma is further covered by the upper lid. From the surgeon's point of view the result looks less pretty after iridectomy; but need we consider this?

THE REMOVAL OF CORTEX AND THE TOILET OF THE WOUND.

The irrigator invented by MacKeown, of Belfast, and described by him in his interesting and instructive work on the "Treatment of Immature Cataract," has been used by me over 800 times for mature and immature cataracts. It is sufficient to remove the nucleus of the opaque lens by expression, and all else can be easily and safely effected by irrigation. Cortex of all kinds, whether flocculent, brittle, doughy, or glutinous, can be washed out *in toto*; the chamber is cleansed by means of a flow of sterilised saline solution, of blood, of *débris*, and of accidentally introduced and septic matter; lastly, the iris is replaced with an ease, certainty, and safety that has no equal in instrumental interference.

On the third day a large proportion of the eyes show a clear black pupil, and secondary cataract is much less common than it is with the ordinary methods of clearing the chamber.

I find that in 250 consecutive cases (in only one of which irrigation was omitted) capsulotomy was called for in nine for the results of cortical matter left behind, and in another series of 500, 460 of which were irrigated, only eleven required capsulotomy.

The even hydrostatic pressure of the aseptic fluid has the additional advantage over other methods that it is attended with a very low vitreous-casualty; the reasons for this are self-obvious. I find that in a series of 500 cases with 460 irrigations, vitreous escape only occurred in ten cases (or in 2 per cent.), while in the 250-series the actual escapes fell to three (or 1.2 per cent.).

When care is taken to sterilise the solution used, no fear of introducing a septic element need be entertained, but it will be obvious that if simple and easily carried-out measures are neglected, the gravest consequences may follow.

ANTISEPSIS AND ASEPSIS.

Few surgeons will disagree as to the advisability of securely sterilising all instruments *save the knife*

and needle by boiling, or better still, by steam. The two instruments named are harder to sterilise because their points and edges are so easily damaged, whereas it is *essential* that they should be as sharp as possible. I have tried boiling and steam (most carefully applied), carbolic lotion (1:40), absolute alcohol, and a variety of other antiseptics, and I find that every one of them damages these sensitive instruments to such an extent as to introduce a definite element of danger into the operation. In my last 800 cases I have used a method of cleaning the knife and needle founded on some unpublished experiments kindly communicated to me by a friend. This gentleman took a Weiss's knife fresh from a cataract extraction, and after washing it carefully in sterilised water, rubbed it clean with sterilised absorbent wool; he then endeavoured to obtain cultivations from the blade, with the result that he found his sterilisation had been most satisfactory. Acting on this I cleanse my knife carefully after each operation with chinisol solution (1:3000), and then wipe it dry with sterilised absorbent wool. The needle is similarly treated. The results have been very satisfactory. In 250 cases there were five septic cases (2 per cent.), in four of which perception of light alone remained, whilst in one vision was lost. A careful study of the cases renders it improbable that the infection was in any case due to the knife, there being other more likely sources of the mischief. Those of us who have done much cataract work in India know how hard it is to keep down our septic list amongst a people who not infrequently deliberately raise their bandages to rub the recently operated eye, or to show it to a friend. It has not seldom been my lot to have all my bandages removed by a foolish patient. One man, in spite of all remonstrance, removed every stitch of clothing, including his bandages, and sat under the tap, as regularly as the sun rose; he did excellently in spite of this, but such vagaries render the attainment of a perfect asepsis highly hypothetical in the East.

For the eye itself and for irrigation of the conjunctival sac I use chinisol solution (1:3000), made from boiled water, but I lay more stress on the asepticity than on the antiseptic action of my solutions, for I have been led to gravely doubt whether one does not do more harm than good by striving to antisepticise a membrane which is so intolerant of interference as the conjunctiva.

MARKING OF THE TONGUE AN EARLY SYMPTOM OF ANKYLOSTOMIASIS.

By A. B. DUPREY, M.R.C.S., L.R.C.P.

Colonial Assistant Surgeon, St. Lucia; late District Medical Officer, Grenada.

THE peculiar marking of the tongue in ankylostomiasis described by Dr. Delamere in the issue of June 16th of the JOURNAL OF TROPICAL MEDICINE is a very common occurrence among immigrant coolies of nearly all ages. I have had frequent opportunities of noticing these marks on their tongues, but never thought of associating them with the condition known as ankylostomiasis. On the contrary,

I have always ascribed these markings to a totally different origin, namely, a staining of the mucous membrane of the tongue, due to a favourite food which all coolies from India, young and old, are in the habit of using, when not only their tongues become discoloured, but their teeth are invariably stained of a brownish or of a purely black colour. A coolie on being asked why it is their tongues and teeth are always more or less discoloured, stated that it was due to their habit of eating the fruit and chewing the leaves of a particular plant, the name of which is pan or phan. This pan or phan is a creeper, and is very common in the West Indies, being usually found wherever there have been immigrant coolies, who, I believe, originally introduced the plant. The creeper bears a fruit varying in size according to the species, and is somewhat pear-shaped. The larger fruit generally tapers below in a long and round-pointed apex. Its outer surface is tuberculated, the tubercles being smooth and triangular, and are arranged in parallel longitudinal columns, a narrow smooth groove intervening between each column. The fruit, when ripe, is of a bright yellow colour, and on section discloses a red pulp; the pips are round and red and lie embedded in meshes of the pulp, somewhat after the style of a water-melon. It has a sweetish, nauseous taste. This fruit when eaten stains the mucous membrane of the tongue and the teeth a yellowish-red, which subsequently turns the tongue and teeth of a brownish or blue-black colour. The leaves are bitter or are said to be so, and are chewed for its supposed preserving virtues. The marking of the tongue I really believe to be due to this phan, and I presume that the reason why it is seen in patches and streaks is that the brown stains, which are not permanent, fade away in a somewhat irregular manner.

The condition recognised as ankylostomiasis is an exceedingly common one in the West Indies, and are chiefly to be found among the native children and youths of the labouring classes. It is mostly due to the inveterate habit of eating dirt or other filth, such as rags, paper, slate, ashes, finger-nails, &c. Such instances of perverted appetites are to be met with commonly in all the islands. In Grenada there is a red-earth which the children are particularly fond of eating, and which is said to possess a somewhat sweetish taste. The anæmia brought on by this habit is a perfectly hopeless condition to deal with, so that positively "They pale, sicken and die." It is the same condition known under the name of ankylostomiasis, but their tongues do not present the markings so ably described by Dr. Delamere. If these brown and blue-black stains should really be an early sign of ankylostomiasis, then I submit that in both classes of patients, viz., coolies and natives alike, the markings should be present, for in that case it would prove beyond a doubt that the marks are not produced by the juices of this phan which the natives of India alone are so fond of chewing.

I have seen coolies in the Windward Islands who may be said to be more "creolised" than those, for instance, of Trinidad or British Guiana, where immigration still exists, and who are not actual dirt-eaters, but whose condition of anæmia is brought about by

a habit of eating raw rice. It is significant that this habit is acquired in the process of washing the rice, which the coolies do thoroughly, going through several washings until the rice is perfectly white. While thus engaged, half a handful of the clean rice is thrown into the mouth and eaten, and thus the habit of raw rice eating is imperceptibly acquired.

In watching this process as I have done, one would think that any ova of the ankylostomes which may be sticking to the grains of rice are washed away, but it is possible that while the habit strengthens, less care is taken of the cleansing process, and the grave consequences of ankylostomiasis result. These "creolised" coolies, being more in touch with the native labourers, have long lost the habit of chewing the phan, hence their tongues and teeth are not discoloured.

I have elsewhere described a very early sign of the condition known as ankylostomiasis, and one which may be detected before any palpable symptom of anæmia develops itself, namely, commencing atrophy of the skin consequent upon a condition of general innutrition. This symptom is not very surprising when one comes to think of it, for it is one which would naturally be expected from a knowledge of the pathology of the condition. Any one who has performed *post-mortem* examinations on subjects of ankylostomiasis cannot help observing the general innutrition and degeneration of the organs and tissues. In the living patient the skin shows this condition of innutrition in a very early stage of the disease.

One would naturally suspect ankylostomiasis in a coolie who comes complaining of palpitation and shortness of breath, though no other sign of anæmia be apparent, since one knows that almost all coolies are geophagists; but take a native child whose mother says is suffering from "Heart-beating and quick breath," and in whom no symptom of anæmia or other disease can be detected, the early sign of innutrition of the skin becomes a valuable means of helping to a diagnosis. In fact, just as the nature of the pulse furnishes us with a valuable index to the working condition of the circulation and other internal derangements, so do the innutrition and peculiar loss of firmness and tone observed in the skin in early ankylostomiasis lead us to form a fairly accurate idea as to the state of the organs and tissues, thus suggesting other means of examination in order to arrive at the true nature of the disease.

SOME UNUSUAL FACTS DISCOVERED DURING THE EXAMINATION OF MALARIAL BLOOD.—G. B. Mariotti-Bianchi, while examining the blood of a patient who had never previously had an attack of malaria, found that abnormal conditions prevailed. The annular form of the parasite was present, and also a few crescents, but, in addition, adult forms were seen in the circulating blood. The author also had the unusual experience of actually witnessing the rupture of a blood-corpuscle and the exit of the crescentic forms.—*La Riforma Medica*, July 12th, 1902.

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THE Journal of Tropical Medicine

SEPTEMBER 1, 1902.

British Medical Association.

SECTION OF TROPICAL DISEASES.

THIRD NOTICE.

Friday, August 1.

YELLOW FEVER.

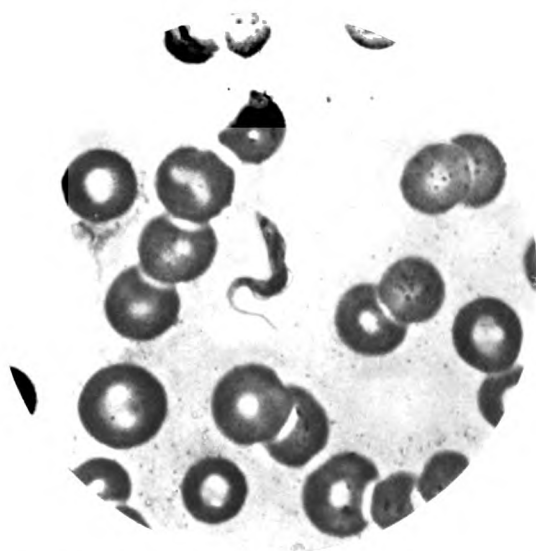
I.—JAMES CANTLIE, M.B., F.R.C.S., Surgeon Seamen's Hospital, London.

IN opening the discussion on yellow fever Mr. Cantlie drew attention to the unsatisfactory term "yellow" as applied to any fever. So many ailments were attended by fever and "yellowness" that a more scientific nomenclature appeared advisable. He suggested "hæmatogenous jaundice."

Attention was drawn to the excellent work done in Cuba recently by the U.S. Army Commission in connection with the etiology and epidemiology of yellow fever. The result of these investigations were:

(1) That no specific bacterium or protozoan of any kind was present in the blood, tissues, or excreta of yellow fever patients.

(2) That the blood of yellow fever patients injected into healthy (non-immune) persons caused an attack of yellow fever. The specific agent is therefore in the blood.



TRYPANOSOMA FOUND IN THE BLOOD OF A EUROPEAN.

Illustrating the article on Trypanosoma in Man, by R. M. FORDE, L.R.C.P., L.R.C.S.,
Colonial Surgeon, Gambia, West Africa.

(3) Blood taken from a yellow fever patient and defibrinated, or diluted and passed through a Berkefeld filter, and the remaining serum injected into the veins of a healthy person caused yellow fever. The specific agent is therefore in the blood serum, and is either a germ which is ultra-microscopic, which cannot be excluded by a Berkefeld filter, or it is non-bacterial and in all probability a toxin.

(4) Yellow fever is not spread by fomites. This was definitely tested for several weeks by allowing healthy (non-immune) persons to sleep on mattresses and pillows and in pyjamas and under sheets and blankets soaked in yellow fever excreta and vomit. None of the persons so exposed contracted yellow fever.

(5) The mosquito—*Stegomyia fasciata*—by its bite conveys yellow fever from the sick to the healthy. This was ascertained by allowing mosquitoes which had fed on yellow fever patients to bite healthy persons, when yellow fever appeared amongst them after certain definite periods. In the room in which the experiment was made other persons protected by a mosquito-proof netting slept, and in no instance did the disease spread to others.

(6) The infected mosquitoes could not inoculate healthy persons with yellow fever until twelve days after they had become infected. This fact would point to a germ rather than a toxin as being the specific agent in yellow fever.

Mr. Cantlie drew attention to the fact that many other diseases such as blackwater fever, bilious remittent fever, bilious typhoid, &c., present many signs and symptoms akin to yellow fever. In Egypt, during the Napoleonic occupation, Professor Sandwith states that the "typhus bilieux" existing in Egypt was designated a variety of yellow fever. It is possible, therefore, that yellow fever exists much more widely than at present believed.

II.—GEORGE C. Low, M.B., C.M.Edin., Cragg's Research Scholar, London School of Tropical Medicine.

THE DIFFERENTIAL DIAGNOSIS OF YELLOW FEVER AND MALIGNANT MALARIA.

Clinically there is difficulty in diagnosing yellow fever from pernicious forms of malarial fever. They have many signs and symptoms in common, notably: The facial appearance; the condition of the tongue and skin; the temperature and its relations to the pulse; albuminuria; the epigastric pain and vomiting; hæmaturia; the nervous symptoms. As a means of ultimate diagnosis one has to rely on the microscope. *Post mortem* the diagnosis is simple: malaria is at once recognised by the presence of malarial pigment, by the recognition of the characteristic parasites in the brain, spleen or gastric mucosa. It is in the early stages of an epidemic that appeal must be made to the microscope; for later on, when the epidemic is at its height, the rapid spread, the high death-rate, and the persistent occurrence of groups of symptoms in a large number of cases from the same area, indicate clearly that yellow fever is present.

DISCUSSION.

PATRICK MANSON, C.M.G., F.R.S., LL.D., warned observers against coming to too hasty a conclusion

from the experiments just published by the U.S. Army Commission. There might be, although he did not say there were, flaws in their methods of investigation, and markedly in the matter of the extent of their researches in regard to the absence of a germ of any kind in the filtered blood of yellow fever patients; he observed that the blood experimented with had been drawn from one patient only. This was liable to beget fallacy.

L. W. SAMBON, M.D. (Naples) thought that the diagnosis between yellow fever and malaria will be more simple when blackwater fever is separated finally from the intermittent fevers with which it is at present associated. He pointed out that in blackwater fever the vomit is hæmatogenous, the jaundice early, and hæmoglobinuria prevails—conditions which do not obtain in yellow fever. The existence of blackwater fever on the West Coast of Africa, Dr. Sambon believes, is largely responsible for the idea that yellow fever ever prevailed there.

W. T. PROUT, M.D.Lond. (Sierra Leone), said that neither his predecessor in office on the West Coast, nor in his own experience, had he ever seen a case of yellow fever on the West African coast. In all probability if the disease ever occurred there the cases were imported thither from the West Indies.

MALTA FEVER.

I.—P. W. BASSETT-SMITH, R.N., Lecturer on Tropical Diseases, Haslar, sent a paper on:—

"The agglutinating properties in the blood in cases of Mediterranean Fever, with special regard to prognosis; remarks on other blood changes and reactions during the course of the disease."

During the past two and a half years 196 cases of Malta fever were admitted to the Royal Naval Hospital, Haslar.

The agglutination of the micrococcus melitensis, when brought in contact with dilutions of blood of patients suffering from Malta fever, is more marked than in the case of typhoid, as it commences on the fifth day and endures at times to one and a half years.

From observations made by Dr. Bassett-Smith it appears that in acute, chronic and convalescent cases, the bactericidal power of the serum against the specific organism of Malta fever is very slight, very much less than that usually of healthy individuals who have never suffered from the disease. If this procedure gives any adequate estimation of the immunity of the subject, then those who have lately recovered from the disease are less able to resist the invasion of the organism if introduced, and therefore more prone for a period to re-infection. If this is so, men who have lately recovered from an attack should not return to the endemic area for some time. The necessity also for early invaliding becomes apparent. In thirteen cases examined, as to the phagocytic power of leucocytes, the average organisms found inside the polymorphonuclear leucocytes were few in cases of Malta fever, whereas in the controls of normal blood they were always higher; it would therefore seem that not only is the bactericidal power of the blood low, but that also the phagocytic properties of the leucocytes are diminished.

II.—BRIAN MELLAND, M.D.Lond.

MALTA FEVER IN THE CANARIES.

In the Canaries the term typhoid fever and gastric fever are both in use; the latter, signifying gastric remittent fever, is but one of the names given to Malta fever. It is doubtful whether malaria exists at all in the Canary Islands. It is only since 1897 that Dr. Melland recognised the prevalent fever of the islands as Malta fever. The disease, however, does not seem so virulent in the Canaries as in the Mediterranean, and in quite 50 per cent. of the cases the fever lasts only two or three weeks. A fourteen-day fever is common, and it is apt to be confounded with acute rheumatism, influenza of the abdominal type, malaria, and abortive or fourteen-day typhoid. Another type is the five-week type; this form may readily be mistaken for typhoid. The long undulant and severe type of Malta fever, with three or more separate attacks separated by intervals of normal temperature, does not exist in the Canaries in more than 10 per cent. of cases.

The mode of spread is obscure; those who have lived long in the endemic area are most likely to contract the disease. The infection is no doubt often inhaled with dust, and many of the country villages and isolated farms seem more dangerous than the paved town of Las Palmas. In the form of the disease met with in the Canaries the spleen is less commonly enlarged; joint pains are rarer. The disease in Canary seems in bad cases to partake of the type of ulcerative colitis.

Treatment.

Liquid diet (milk) should be administered at first; the Spaniards often treated the ailment with purgatives—calomel, $\frac{1}{4}$ grain every eight hours—and a single large dose of ipecacuanha is often useful. Bismuth, salol, benzol naphthol are given six hourly; and the colon is washed out daily with one litre of warm boracic solution.

III.—JAMES A. HISLOP, L.R.C.P., L.R.C.S.Edin.

THE GEOGRAPHICAL DISTRIBUTION OF MALTA FEVER AND THE VALUE OF SPLENIC ENLARGEMENT AS A TEST OF MALARIAL INCIDENCE.

From experiences gathered in Assam, Dr. Hislop comes to the conclusion that Malta fever exists there. Out of eleven cases of fever, selected more for a continuance of fever than for any undulatory character, ten gave a well-marked reaction to Malta fever. In many of the "fever" cases the splenic enlargement was enormous—a condition usually attributed to malaria—but Dr. Hislop is of opinion that the spleen enlarges in "fever" from causes other than malarial—a fever in which quinine has no effect.

IV.—CHARLES A. BENTLEY, M.B., C.M.Edin.

KALA-AZAR AS AN ANALOGOUS DISEASE TO MALTA FEVER.

In this paper Dr. Bentley publishes some "Preliminary Notes of an Investigation, and some Discoveries regarding the Nature of the Condition known as Kala-azar." He has come to the following conclusions:—

(1) Kala-azar is neither malarial fever, benign or malignant; nor is it any form of malarial cachexia, or post-malarial cachexia. It may, however, be complicated by a coincident malarial infection.

(2) Kala-azar is a distinct disease, consisting of an initial attack of irregularly intermittent, remittent or continued fever; this is followed by recurrences of a similar nature in the first stage, by a nearly continuous fever of a low type in the second stage, the whole together constituting a febrile affection of long duration.

The wide divergence between kala-azar and malaria has been frequently noted, and the idea that it is non-malarial in origin is no new one. The atypical periodicity of kala-azar serves to distinguish it from malaria; the high death-rate, the intractability of the disease to quinine, the communicability of kala-azar, the absence of parasites and pigment, and the existence of low fever in the later stages of the disease, serve to distinguish it from malaria.

The serum test afforded positive evidence also to favour Dr. Bentley's opinion that Malta fever is present in the district in Assam in which he practises (the Borjalie Tea Estate, Tezpur, Assam.)

J. EVERETT DUTTON, M.B.Vict., Walter Myers
Fellow, Liverpool School of Tropical Medicine.

NOTE ON A TRYPANOSOME OCCURRING IN THE BLOOD OF MAN.

Dr. Dutton, while staying at Bathurst, Gambia, during 1901, found in the blood of a man just returned from England, having been invalided home six months previously after an attack of fever, a flagellated protozoan belonging to the genus *Trypanosoma*. The subject of the disease had been in the Gambia for seven years, and except for slight malarial attacks had never been ill. In May, 1901, he fell ill, and although he had fever, no malarial parasites were found in his blood. The patient suffering from occasional rise of temperature, pulse, and respiration varied in frequency, the liver and spleen were somewhat enlarged and painful, the heart sounds were weak, and the patient complained of breathlessness and weakness.

The most notable features of the illness have been: (1) Its chronic course; (2) the general wasting and weakness; (3) the irregular rises of temperature, never very high, and of a relapsing type; (4) local œdema; (5) congested areas of the skin; (6) enlargement of the spleen; (7) constant increased frequency of pulse and respiration.

[These signs and symptoms were present when Dr. Dutton showed the patient to members of the Section of Tropical Diseases, B.M.A. when they visited Liverpool on August 1st, 1902.—EDITOR.]

Dr. Dutton proposes to name the parasite which he has discovered the *Trypanosoma gambiense*.

GEO. A. WILLIAMSON, M.D.Aberd., Larnaca, Cyprus.
BILHARZIA HÆMATOBIA IN CYPRUS.

A native of Cyprus, aged 22, who resided in Morphou, in the north-west of the island, was found by Dr. Williamson to be the subject of bilharzia. The patient had never left his native village until eighteen months previously, when he went to Nicosia

and enlisted in the Police. He remained there five months, and was then transferred to Larnaca, where he has resided up to the present time. The man had never left the island of Cyprus. Some six months before admission to hospital the patient began to pass blood, and noticed that the blood appeared with the last drops of urine; he had no pain, nor any difficulty in passing urine. Blood escaped every day, and some mucous discharge was also present when the blood came. The typical bilharzia ova were found in numbers in the urine.

Treatment.

Best in bed and boracic acid and buchu internally. The blood and ova disappeared from the urine, but Dr. Williamson does not anticipate a permanent cure.

Dr. BRUNO GALLI-VALERIO, Professor in the University of Lausanne.

CONTRIBUTION TO THE STUDY OF *BACILLUS PESTIS*: ITS CULTURAL AND MORPHOLOGICAL CHARACTERS AND ITS RELATIONS WITH *BACILLUS PSEUDO-TUBERCULOSIS RODENTII*.

Dr. Galli-Valerio finds that the morphological characters of the *Bacillus pestis* are cultivated at a temperature of 18° to 20° C. At 37° C. the bacillus may not grow on cultures at all, but at + 1° C. and — 5° C. are even slightly developed. Dr. Galli-Valerio describes the characters of the cultures of the *Bacillus pestis* on gelatin, on agar, on agar with glycerine and glucose, on sloped gelatinised ox serum, on sloped gelatinised pleuritic fluid, and on rabbit-serum, on potatoes, milk, broth, &c. He finds that the organism most closely resembling the plague bacillus is the *Bacillus pseudo-tuberculosis rodentium* (guinea-pig).

GEO. A. WILLIAMSON, M.D. Aberd., Larnaca, Cyprus. STATISTICS OF THE BLOOD EXAMINATION IN CASES OF MALARIA IN CYPRUS DURING A PERIOD OF TWELVE MONTHS.

Out of 503 cases of illness diagnosed as malaria in Larnaca, Dr. Williamson, during the period from February 1st, 1901, to January 31st, 1902, found parasites in 470, i.e., 94 per cent. The number of cases in the twelve months commencing February 1st, 1901, and ending Jan. 31st, 1902, were respectively 11, 9, 5, 6, 9, 101, 87, 94, 60, 24, 32, 32, the fewest number of cases occurring in April (5), and the maximum in July (101). The total number of the tertian form of fever amounted to 308, of which 107 were double tertians. The total number of quartan fever was 49, of which 7 were double quartan and 8 triple quartan.

L. W. SAMBON, M.D., London School of Tropical Medicine.

REMARKS CONCERNING THE NOMENCLATURE, ETIOLOGY AND PROPHYLAXIS OF THE INTERMITTENT FEVERS.

An appropriate nomenclature is, according to Dr. Sambon, a necessity. Should a collective name be desired, intermittent fever might be retained or that of hæmocytozoal fever selected. The term malaria might be set aside; æstivo-autumnal is inappropriate. The re-instatement of the ancient names of semi-

tertian and sub-tertian used by Hippocrates and others has much to recommend it. The term "plasmodium" should be given up and the quartan, tertian and sub-tertian (æstivo-autumnal) classed in one genus and called respectively *Hæmamœba Golgi*, *H. Vivax*, and *H. Laverani*.

The fever-producing cycle of the parasite might be called the asexual or swarming stage. The mosquito stage might be termed the sexual or resting stage. Dr. Sambon dealt with the geographical distribution of the hæmocytozoal fevers and suggested a thorough investigation of the distribution of quartan fever.

Dr. Manson thought Dr. Sambon's suggestion a good one. Dr. C. W. Daniels considered a change in nomenclature would probably cause great confusion. Surgeon-Major Poole (I.M.S., retired) said he had seen cases of quartan fever in Bengal.

The British Guiana Medical Annual for 1902.

NOTES ON MALARIA AND OTHER TROPICAL DISEASES DURING THE TOUR OF THE ROYAL COMMISSION ON MALARIA.

C. W. DANIELS, M.B. Cantab.

Medical Superintendent London School of Tropical Medicine.

It has been suggested by the editors of the *British Guiana Medical Annual* that a summary of the observations made in the course of my investigations in India and Central Africa might be of interest.

The main result was the complete corroboration of what, when I left British Guiana in 1898, was considered to be a mere hypothesis, the malaria mosquito theory. Thanks to Major Ross, I.M.S., I was able almost immediately after landing in Calcutta to observe the complete development of the *Proteosoma*, a bird parasite closely resembling the human benign parasite, in the mosquitoes, and was shown by him specimens, of the developed human parasite in a mosquito's stomach, thus leaving little doubt remaining as to the similarity in the process of infection of man and birds by the malarial parasite and the *Proteosoma* respectively. The development of the human malarial parasites we were unable to trace though several species of mosquitoes were tried. One of these was an anopheles, and specimens sent home of this mosquito were described and named by Giles as *Anopheles Rossii*.

The weather was cold and we could not satisfy ourselves that climatic conditions were not in part responsible for our failure; but subsequently other observers have shown that this anopheles, even under favourable conditions, does not carry malaria.

In Africa one was at once struck by the large number of anopheles present and the rough general correspondence between the numbers of these mosquitoes and the liability to early infection with malaria.

Direct experiments to show which mosquitoes carried human malaria and the stages of development of the parasites were much delayed, as so few of the

cases of malaria developed crescents in large numbers. If repeated examinations are made, over 70 per cent. alike of Europeans and natives, children and adults, after an attack of fever are found to have "crescents," but usually in very small numbers, one being found after prolonged search on one day, and perhaps for the next two or three days these examinations are negative. On numerous such cases I fed mosquitoes, but in no case were they infected. In May, 1899, out of four mosquitoes (*Anopheles funestus*) fed on a moderate "crescent" case, one was infected, but I had no further success till in November and December in the same year, when I had a patient with numerous crescents, and from him I infected twenty-seven of these mosquitoes and was able to trace the full development of the parasites. The mosquito used had, in August, 1899, been shown by the first Liverpool Malaria Expedition to carry the parasite of quartan malarial fever, as one out of five fed on a patient with quartan parasites developed zygotes.

Some time was spent in determining the breeding places of mosquitoes in general, and particularly those of the genus *Anopheles* and *Anopheles funestus*. It was found that the permanent and constant breeding place was running water, and particularly the grass-grown shoals in and at the edge of rivers. In other places they were found only during the rainy season, or when the level of the subsoil water was high, or in a few large collections of water. Small collections soon become too stagnant for *Anopheles funestus* and some other *anopheles* to develop. These results explained well the local distribution of malaria in British Central Africa, but it was obvious that with different conditions as to rainfall and the character of the soil there would be a considerable alteration in the conditions, and that places which were of little importance in British Central Africa might with the heavier rainfall and waterlogged soil of places like British Guiana be of the greatest importance. The fairly frequent occurrence of *anopheles* larvæ in rivers has been discovered by Nuttall in England quite independently, but elsewhere this class of breeding place has received little attention. The absence of rivers in Barbados is the probable explanation of the absence of *anopheles* there.

As I had anticipated, the great majority of cases of malaria amongst the natives occurred in children. *Post-mortem* examinations were not possible, so that a repetition of my observations in British Guiana on the proportion at various ages with malarial pigment in the organs was impracticable. As an alternative I determined the number with enlarged spleens with the result that in the most malarial district no less than 68 per cent. of children under 2 years of age were found to have markedly enlarged spleens. In a less malarial district the proportion was much lower under 4, but higher over 4. The results in three districts are embodied in the subjoined table:—

| | Highlands, 3,000 ft. or more | Level of Lake Nyassa, 1,500 ft. and under | Lower Shire River, 300 ft. or less |
|---------------------------|------------------------------------|--|--|
| Aged 2 years and under .. | 18.4 | 68.8 | 48.1 |
| " 2-4 " .. | 31.5 | 45.1 | 57.1 |
| " 15 " and under .. | 26.2 | 11.5 | 13.7 |

Numbers give the percentage of persons at the ages specified with marked splenic enlargement.

Malaria in the great majority of cases was due to the autumnal parasite. The parasites were found in all cases of this fever, and on several occasions in apparently healthy native children, but even amongst these close enquiry would often show that there had been some periodical symptoms, and there was evidence, not only that splenic enlargement resulted from these infections, but that death from pernicious access was fairly common. The more intensely malarial the district the higher was the mortality in childhood.

The incidence of malaria on children and the freedom of adults was more absolute than is the case in natives of British Guiana, probably because even the least malarial districts seem to be more malarial than the British Guiana littoral. In Georgetown it was fairly common for a newcomer to go two years or more without getting his first attack of fever. In the Central African Highlands the first attack is usually in a year, whilst in the most unhealthy districts few persons are resident for a month without getting an attack. As about a fortnight is required for incubation this would mean that, whilst in the most unhealthy parts of Central Africa fourteen days' exposure is nearly certain to mean malarial infection, two years is required in Georgetown for at all certain infection. In other words, the liability to malarial infection in some parts of Central Africa (Upper Shire Valley) is more than forty-eight times as great as in Georgetown.

Nothing of any great importance was found as regards blackwater fever. Many of the cases of this disease are mild and the great danger is suppression of urine. Malarial parasites are not found during the attack either in the blood or in fatal cases in the organs. In all the cases I saw there had been malaria immediately before. No drugs appeared to have any specific action. Quinine, even in large doses, did not affect either favourably or otherwise the course of the disease. I saw nothing which convinced me that quinine had anything to do with the causation of blackwater fever. In some cases no quinine had been taken, and in the others there was no definite relation between the amount taken or the time it was taken and the time of the onset or the severity of the disease.

In England as regards some points malaria is more readily studied than in the Tropics. The cases are not very numerous but can be more closely watched. The effects of the surroundings on the patient and his parasites are not complicated by the possibility of a fresh infection. With both tertian and quartan parasites symptoms occur in accordance with the number of parasites present. In many cases rest in a warm bed without treatment results in sufficient diminution of the parasites, so that no symptoms, *i.e.*, no fever, occur. This condition can persist for an indefinite period, parasites being constantly present in small numbers without any fever or any tendency to a great increase in the numbers of the parasites. In such a patient, however, exposure to cold in some way enables the parasites to increase sufficiently to cause fever. The number of parasites necessary to cause

symptoms appears to be for most patients in England about 200-300 per cubic millimetre of blood.

During the summer of 1901 I paid a short visit to Sierra Leone. At that time there was very little fever there, but I was shown specimens of quartan and tertian as well as the autumnal-parasite by Dr. Prout.

The conditions as regards breeding grounds of anopheles were very different from those in Central and East Africa. The rocky nature of the soil and the heavy rainfall which during the period I was there occurred daily, result in the formation of rock pools and pools in hollows which neither become dry nor stagnant, and in these anopheles larvæ breed abundantly.

Major Ross and Dr. Taylor had commenced destroying these places, and had made such progress as to demonstrate the practicability of rendering that town free from anopheles during the wet season. The expense was considerable, but not out of proportion with that of the benefits to be derived, even on pecuniary grounds alone.

In the course of my wanderings I have not seen in any place any outbreak or attack of malaria which could not be readily accounted for by the now well-known facts that the gametes or sexual forms of the malarial parasite develop when taken with the blood into the mosquito's stomach and are finally injected by the mosquito into other men and after a variable period, usually one to three weeks, give rise to a sufficiently large malarial infection to cause "fever."

As far as I have observed there is no evidence of any other mode of transmission, but careful and prolonged observations are necessary to absolutely prove a negative.

It may, however, be mentioned that in other branches of medicine, such as the development of tape-worms, when one method of infection is proved and explains all known facts about that tape-worm, action is taken for practical purposes as if that were the only important method, and I consider that malaria should be dealt with on the same lines and the known causes diminished or removed.

Attention in India and elsewhere has at length been drawn to the necessity of a critical examination of the value of enlargement of the spleen as a test of malaria, with the result that conclusions similar to those drawn in the *British Guiana Medical Annual* for 1895, from a comparison of the ages of incidence of enlarged spleen and malarial pigmentation of the organs, have been shown to hold good for the ages of incidence of enlarged spleen and the ages at which malarial parasites are most commonly present.

Malaria is most prevalent in childhood and diminishes in frequency in adults who have been exposed to malarial infection from childhood. Enlarged spleens amongst Indians, on the other hand, are most abundant at periods of life when malaria is rare.

This conclusion does not hold good for negroes; enlarged spleen and malarial parasites are alike common in childhood and rare in adult life.

Of diseases other than malaria also met with in India, Central Africa, East and West Africa, filariasis has met with most attention. The *F. nocturna* has been shown to be carried by some species of anopheles

and panopletes as well as culex, but the discovery of importance is that by Low in England, and subsequently, but independently, by James in India, that Bancroft, junr.'s suggestion that the filarial embryos escaped through the proboscis and not, as was previously supposed, into water from the body of the mosquito is the correct one. Prophylaxis on lines similar to that for malaria is therefore requisite.

F. perstans has been shown to be steadily extending across Africa from west to east, and "sleeping sickness" is extending in a similar manner. It is of the utmost importance that it should be conclusively shown either that this disease does or does not occur amongst the aboriginal inhabitants of British Guiana.

Loos' discovery that the anchylostome embryo can penetrate the epidermis is a most important one. His sections leave no doubt that this occurs, and his discovery has been confirmed in India, and also on guinea-pigs in Liverpool. The proof that having penetrated the skin the anchylostome is able to reach the intestine is not conclusive.

That the anchylostome embryos cause an eruption, probably the "ground itch," of British Guiana, seems to be established.

The granuloma of the pudenda, groin ulceration of British Guiana occurs on the West Coast of Africa, and after inspection of cases of madura foot, mycetoma, I can readily recall cases of that disease which I have seen in British Guiana.

Of diseases I had not previously seen in British Guiana, plague, cholera, sleeping sickness, blackwater fever, goundou and Malta fever, are of the most importance. Though the advances in the last few years have been great much remains to be done. Little more is known about dysentery than was the case a few years ago, and the part played by the *amœba coli* is undetermined, and the life-history of that protozoon not thoroughly known. Of parasites allied to human malaria, the *Halteridium*, the commonest of all *Hæmosporidia*, has not been worked out, and there is strong negative evidence that it is not carried by mosquitoes.

F. nocturna and *F. inmitis* are carried by mosquitoes, though there are differences in detail, but the carriers of *F. perstans*, *F. Ozzardi*, *F. Demarquayi* and *F. diurna* are not known.

LIFE-HISTORY OF THE ANKYLOSTOMUM DUODENALE.

By A. T. OZZARD, M.R.C.S.

DURING the last two years I have been able to completely confirm the experiments of Lieut.-Colonel Giles with regard to the life-history of this parasite. That this is an example of *dimorphism* or *heterogenesis*, I have not the slightest doubt.

I conducted my experiments on exactly similar lines to those described by Colonel Giles.

A small quantity of the dejecta, known to contain only the ova of ankylostomum of duodenale, is mixed with about three or four times its bulk of water, and poured on the surface of some sterilised river sand in a suitable vessel. The latter is covered over with a piece of glass. Every day a small portion of the

fæcal layer was placed on a glass slide, a smear preparation made, and examined under a low power of the microscope.

At the ordinary temperature of the air here, the embryos usually hatched out within twenty-four hours. During the rainy season, however, when the temperature is lower, they sometimes take a week before hatching out.

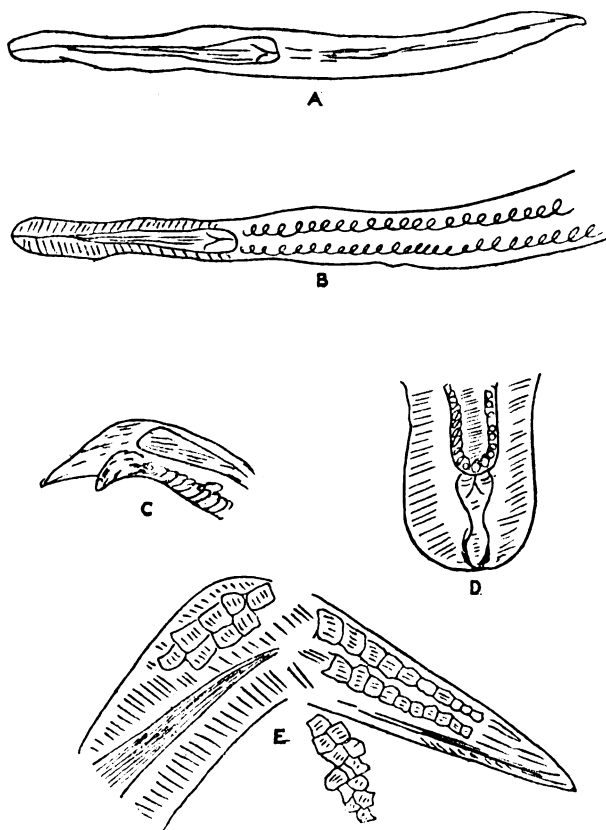
In emerging from the egg the embryo measures roughly from $\frac{1}{11}$ to $\frac{1}{10}$ in. in length, by $\frac{1}{80}$ in. breadth. At this stage they show little else than an intestine and œsophageal bulb. They now increase rapidly in size and length and undergo numerous ecdyses. Giles says that ecdysis takes place roughly

who will exercise a reasonable amount of trouble over the matter.

The two worms are quite distinct. In the free female forms of *ankylostomum duodenale* the ova are arranged in a single row and of an oval shape, usually from six to ten in number; whereas in the female *Rhabdonema intestinale* the ova are arranged in a double row and are somewhat rhomboidal in shape, with the exception of the lower six or so which are arranged in a single row and appear as if united in a chain.

Anguillula stercoralis develops only in foul water, and rapidly dies in decomposing fæces. The rhabdites of *ankylostoma* mature rapidly and best in the decomposing fæcal matter which is their natural habitat (Giles). I tried several times to cultivate the *ankylostomum duodenale* in water, but invariably failed.

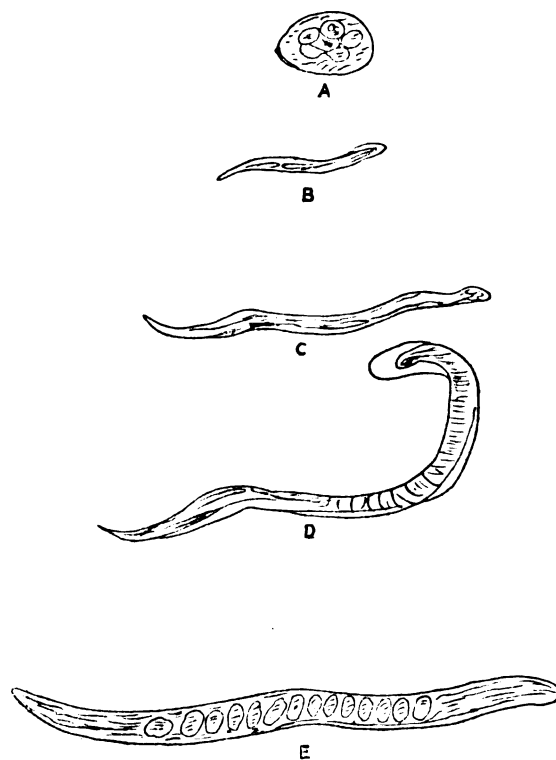
Anguillula stercoralis comes to maturity in a very short space of time compared with that of *ankylos-*



RHABDONEMA INTESTINALE. A, Male; length of gullet = nearly $\frac{1}{2}$ whole length of body. B, Female; length of gullet = nearly $\frac{1}{4}$ whole length of body. C, Tail of male. D, Head of male. E, Adult female, with ova.

about twice in the twenty-four hours. And, as far as I could make out, this represents the case very fairly. The embryos developed into free sexually mature worms in from eight to ten days in my cases. The eggs in the free female worms are exactly similar in size and shape to those of the parasitic form. The size and shape of the *free sexually-mature worms* are, however, very different to those of the parasitic form: being very much smaller generally.

That there can be any confusion between the free sexually-mature forms of *ankylostomum duodenale* and *Rhabdonema intestinale* is impossible to any one



ANKYLOSTOMUM DUODENALE (Free Stage). A, Ovum (segmentation). B, Embryo (2nd to 3rd day). C, Embryo (4th to 6th day). D, Embryo (7th to 8th day). E, Fully-developed female form (8th to 10th day).

toma. The former, in my experiments, developed within two to three days; whereas *ankylostoma* took at least eight to ten days, under the same conditions.

The size of the free adult *ankylostoma duodenale* in my specimens (dried, stained, ones) was roughly about $\frac{1}{7}$ in. in length, by about $\frac{1}{50}$ in. in breadth.

The diagrams are rough sketches from actual specimens in my possession, and are not mere reproductions from other drawings.

ANKYLOSTOMIASIS: THE CAUSE OF THE ANÆMIA IN.

By A. T. OZZARD, M.R.C.S.

It will be noticed that Leonard Rogers, in his definition of ankylostomiasis, assigns as the cause of the anæmia produced "the long-continued small losses of blood through the gastro-intestinal mucous membrane caused by the presence of several hundreds of ankylostoma acting for many months."

Daniels and others, however, suggest that the anæmia is really produced by means of certain toxins elaborated by the ankylostoma.

Personally, I am inclined to agree with Rogers that the anæmia is caused by the actual loss of blood from the bites of the parasites. It is quite true that one ankylostome can imbibe but a very small quantity of blood; but the wound having once been inflicted in the mucous membrane may continue to ooze blood for some time subsequently to the falling off of the begorged parasite, and when it is a question of some hundreds of these parasites acting in a similar manner, I do not think it unreasonable to suppose that a considerable degree of anæmia should be produced thereby in the host.

An important point which appears to me to be in favour of the anæmia being caused by the loss of blood from the bites of the ankylostoma is this: that it is exceedingly remarkable to notice how very rapidly a return to normal is made when treatment with thymol has been successfully carried out. A patient whose mucous membranes are pearly white on admission to Hospital, a few days after a course of thymol will (provided the anæmia is not too far advanced) show an extremely rapid return to the healthy reddish appearance which the mucous membranes should naturally possess. Now if the anæmia were due to certain toxins, the action of which on the blood would, I presume, be to cause a destruction or *poikilosity* of the corpuscles, I maintain that a return to a normal condition of the blood would involve a considerably longer period of time than that seen when thymol has been successfully administered.

Current Literature.

BLACKWATER FEVER AND MALARIA.—In this article C. W. Schlayer cites an interesting case in which the hæmoglobinuria was due, not to quinine, which the patient had taken with impunity during his stay in Africa, but to phenacetin, 0.75 gramme of which was taken. Owing to pleurisy being present, the diagnosis at first presented some difficulty. That the typical attack of blackwater fever was due to the phenacetin is proved by the disease setting in twenty-four hours after its administration, whereas no quinine had been taken for ten days previously. During the attack the treatment consisted in 0.5 gramme of quinine every three hours, and the results were highly satisfactory. This case was an instance of phenacetin-hæmoglobinuria—a very rare condition.—*Deutsche Med. Wochenschr.*, July 10th, 1902.

BLACKWATER FEVER.—The author of this paper, Dr. Reinhold Ruge, gives some valuable hints as to

the treatment of blackwater fever. He asserts that quinine enemata as recommended by Kleine are advantageous in their effect on the general condition of the patient. He is of opinion that the quinine prophylaxis is by no means always a preventive of blackwater fever, and he cites a case in which 3 grains of quinine caused an acute attack of hæmoglobinuria. An impending attack of blackwater fever may be foreshadowed by a microscopical examination of the blood, which twenty-four hours before the attack exhibits polychromatophilic degeneration, with many macrocytes, microcytes, and "blood-shadows."—*Deutsche Med. Wochenschr.*, July 10th, 1902.

MALARIA AND CANCER.—Statistics from Madeira are furnished by J. Goldschmidt with the view of contradicting Loeffler's theory that malaria and cancer do not co-exist in the same place. In the island of Madeira malaria is practically unknown, and carcinoma is of rare occurrence. It is strange in view of Loeffler's theory that carcinoma is not exhibited in a greater proportion in the population of this country.—*Deutsche Med. Wochenschr.*, July 10th, 1902.

THE MECHANISM OF INTESTINAL INFECTION IN DYSENTERY.—According to L. E. Bertrand, the intestine, under certain conditions, is a closed vessel, and favourable to the production of dysentery. According to Virchow, rectitis due to constipation is the chief cause of dysentery, the intestine being practically closed. When opium and astringents are administered during the initial stages of an intestinal flux the intestine is prevented getting rid of the mucus, blood, and other contents, and being thereby placed in the condition of a closed tube is in the worst state possible as regards possibility of curing the disease. The only rational treatment is to promote evacuation; all other methods of treatment are accessory.—*Revue de Médecine*, July 10th, 1902.

ACUTE GASTRO-ENTERITIS, OR SUMMER DIARRHŒA OF INFANTS.—Margaret Taylor Shult recommends that if a child vomits frequently the stomach should be washed out, relief being usually afforded after only once doing so; if, however, the vomiting persists, minute doses of calomel will be found useful, as this drug allays the gastric irritation, cleanses out the intestinal canal, and is, moreover, a strong intestinal antiseptic. Rectal enemata are serviceable when the stools are small and malodorous. If nausea is absent, a dose of castor oil, or 1 to 10 grs. of calomel, should be given after flushing out the colon. Only cold boiled water should be given for twelve hours—no milk—to be followed by a liquid preparation of beef peptonoid. Nurses should bear in mind the fact that the ailment is of an infectious nature, and that therefore all the discharges and stools should be disinfected, the hands kept scrupulously cleaned, and the napkins boiled before being again used.—*Journal of the American Medical Association*, August 2nd, 1902.

CHOLERA.—The efforts of the sanitary authorities in the Philippines to suppress the cholera epidemic have been materially aided by typhoons. A second typhoon of the season visited the islands the last two days of July and cleansed Manila thoroughly.

The sea was forced into the canals and flooded the districts where cholera has been raging. The result was that there were only twenty-five deaths from the disease on July 31st, and the number of new cases was greatly reduced.—*Med. Record*, August 9th, 1902.

PLAGUE.

PREVALENCE OF THE DISEASE.

INDIA.—The deaths from plague in India during the weeks ending July 26th and August 2nd numbered 1,459 and 1,902 respectively. The minimum mortality from plague for the current year seems to have been reached during the weeks ending July 12th and 19th, when the mortality from plague was recorded as 1,058 and 1,100 respectively. The disease is diminishing in the Punjab and increasing in the Bombay districts.

EGYPT.—During the weeks ending August 10th and 17th the fresh cases of plague in Egypt amounted to 5 and 3, and the deaths from the disease to 1 and 1, respectively. On August 17th 10 cases remained under treatment.

CAPE OF GOOD HOPE.—At Port Elizabeth, during the weeks ending July 20th and August 2nd, the fresh cases of plague were reported as 2 and 0, and the deaths from the disease 0 and 1, respectively.

HONG KONG.—In Hong Kong the fresh cases of plague returned during the weeks ending August 16th and 23rd numbered 12 and 12, and the deaths from the disease as 11 and 11, respectively.

MAURITIUS.—During the three weeks ending August 8th, 15th, and 23rd, 2, 0, and 1 fresh cases of plague were reported in Mauritius, and 2, 1, and 1 deaths from the disease, respectively.

QUEENSLAND.—The last case of plague occurred in Queensland on May 31st, 1902. During the outbreak of the disease the total cases of plague reported amounted to 81, of which number 25 died.

CHOLERA IN EGYPT.

DURING the weeks ending August 11th and 18th the cases of cholera reported in Egypt numbered 238 and 1,127 respectively. The deaths from the disease numbered 186 during the week ending August 11th, and 791 during the week ending August 18th. Although 150 cases were reported during the latter week in Cairo, a large number of these are really in villages outside the city itself. During the week ending August 18th 72 new localities have become infected by cholera. The chief outbreaks are in villages which depend entirely for their water supply on surface wells.

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Médecine Navale.
Archives Russes de Pathologie, de Médec. Clinique et de Bacteriologie.
Australasian Medical Gazette.
Boletín de Medicina Naval.
Boston Medical and Surgical Journal.

Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Brooklyn Medical Journal.
Caducée.
Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Esercito.
Hong Kong Telegraph.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
Journal of the American Medical Association.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal
Medical Brief.
Medical Missionary Journal.
Medical Record.
Medical Review.
Merck's Archives.
New York Medical Journal.
New York Post-Graduate.
Pacific Medical Journal.
Polyclinic.
Public Health.
Revista de Medicina Tropical.
Revista Medica de S. Paulo.
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The Journal of Tropical Medicine.

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Original Communications.

REMARKS ON THE NOMENCLATURE, ETIOLOGY AND PROPHYLAXIS OF THE INTERMITTENT FEVERS.¹

By LOUIS W. SAMBON, M.D. (Naples).
Lecturer to the London School of Tropical Medicine.

NOMENCLATURE.

OUR knowledge concerning the etiology of the intermittent fevers has attained such a degree of exactness, that I think it is time we should reject from the nomenclature applied to these fevers those terms which express ideas totally at variance with the ascertained facts. The need of an appropriate terminology is so greatly felt that new names are constantly being suggested both by physicians and zoologists.

The Italian term *MALARIA*, which means noxious air, is the one now most generally used in English medical literature to denote the various intermittent fevers collectively. It was possibly suggested by the ancient term *Mephitis*, name of an old Roman deity which personified noxious effluvia. I think it would be advisable to abolish it, as the French have done, because it expresses and perpetuates a wrong theory, which is, unfortunately, still prevalent amongst the uninformed. The old popular name *AGUE*, from the French term *aigu*, which means acute, has already gone out of use. *PALUDISM* is another foreign term occasionally met with in English medical literature; it was popularised by Laveran, but had been previously used by other French writers as a synonym of *Fièvres palustres*. Its English equivalent is the obsolete expression, *MARSH MIASMATA*.

Should a collective name be deemed necessary, we might surely continue to use the old appellation of *INTERMITTENT FEVERS*, which expresses the dominant

ing feature of these fevers, namely, their periodicity; or we might call them *HÆMOCYTOZOAL FEVERS*, thus designating by one term their causative agent and its anatomical habitat.

Major R. Ross² proposed the denomination of *GNAT FEVERS*. This is evidently unsuitable, because whilst, so far, only a few species of a single genus of the vast family of *Culicidæ* (gnats) are known to foster and propagate the hæmocytozoa of the genus *Hæmaphysa* in man, other similar parasites of the genus *Piroplasma*, in dogs and cattle, are disseminated by ticks.

The hæmocytozoal fevers have also been called *PERIODIC*, *RHYTHMICAL* and *PAROXYSMAL FEVERS*, but the equivalent appellation "intermittent fevers" is preferable, and has the great advantage of being already very generally used.

Considering the intermittent fevers individually, we find that the ancient names for three of these fevers have been retained throughout medical literature; they are: *QUOTIDIAN*, *TERTIAN* and *QUARTAN*; the other, the *SEMI-TERTIAN* (*ἡμιτερταῖον*) of Hippocrates, of Celsus, of Galen, of almost all the ancient physicians, was dropped long ago, evidently on account of the confusion which prevailed about this more obscure and fatal disease. Colin and Sternberg proposed to call it *REMITTENT FEVER*, but this type of fever is really intermittent and only apparently remittent when the intermission is masked by the protraction of its "hot stage." In 1866, Prof. G. Bacelli proposed to call it *SUB-CONTINUOUS FEVER*. In 1889, Marchiafava and Celli, in publishing their important observations on the parasitology of this disease, suggested the name *ÆSTIVO-AUTUMNAL FEVER*, because they noticed that it prevailed during summer and autumn in the Roman Campagna; but this fever has a very wide distribution, and, in tropical regions, it is not limited to the seasons implied in the above name. Prof. Koch recently proposed the name *TROPICAL MALARIA*, but this appellation is doubly unsuitable.

¹ Read at the Manchester meeting of the British Medical Association, August, 1902.

² Report of the Liverpool Malaria Expedition to West Africa, August, 1899.

The disease, although very prevalent in certain tropical regions, is by no means confined to the Tropics, and the term malaria, for reasons already stated, should be discarded. I think that the ancient name, Semi-Tertian, or better, SUB-TERTIAN, should be reinstated. It is cognate to those of the other intermittent fevers, it indicates the type of fever and at the same time its apparent irregularity, it commends itself on account of its simplicity, and has every right to be adopted in accordance with the accepted rules of scientific nomenclature.

Before proceeding to discuss the nomenclature of the parasites, because Prof. Laveran, and the French school in general, still hold them to be mere interchangeable varieties of a same polymorphous species,¹ I deem it necessary to state that I consider the parasites of quartan, tertian, sub-tertian and quotidian as distinct species. In Italy, in Germany, in the United States of America, and in England, all authorities are now agreed in believing that the parasites of the various types of intermittent fever represent distinct species. Numerous facts in their morphology, life-history, geographical distribution, and pathogenesis strongly support the separate nature of these parasites, but the most convincing and irrefutable proof is that afforded by precise inoculation experiments. In fact, the inoculation of one type of parasite, either by means of the mosquito or by the direct inoculation of human blood from man to man, gives rise to the same type of parasite and to no other in the individual inoculated. I am fully aware that some old, careless experiments were, apparently, in favour of polymorphism, but all recent experiments, when scrupulously carried out, have proved the specific distinction of the hæmamoebidæ of man. Those who still believe that the parasites of the intermittent fevers belong to one pleomorphic species follow in the steps of others who, likewise, from lack of continued observation, denied the existence of species in Bacteria. About fifty years ago it was maintained that all sorts of weeds were produced from the seed of the wheat-plant, and people in other respects well educated and intelligent believed that this was possible, because these weeds sprang up in the spots where wheat had been sown!

The parasites of the intermittent fevers belong to the class *Sporozoa*, and to the order named *Hemosporidia*, by Prof. Mingazzini in 1890, but, with Prof. Laveran, I prefer the term *Hæmocytozoa*, proposed by Danilewsky in 1885.

When Laveran first discovered the parasites of the intermittent fevers, he supposed that there was only one parasite, and that the perfect state of this parasite was represented by the microgamete, which he assigned to the vegetable kingdom, and to the genus *Oscillaria* under the name of *Oscillaria malariae*. But he soon abandoned this view and adopted the very general designation of *Hæmatozoon malariae*.

Numerous other names have been suggested, such as *Hæmatophyllum*, *Hæmatomonas*, *Cystosporon*, *Hæmoproteus*, *Plasmodium*, *Polymitus*, &c., but they never

came into use with the exception of *Plasmodium malariae*, proposed, in 1885, by Marchiafava and Celli, from analogy with *Plasmodiophora brassicae*, an amoeboid endocellular parasite which causes the disease called "club-root," or "fingers and toes," in turnips and cabbages. The term *plasmodium* is perhaps the most inappropriate of all the names suggested for the parasites of the intermittent fevers. A plasmodium is a large, irregular mass of protoplasm containing many nuclei. It is composed of numerous cells which have coalesced and are usually enclosed in a common pellicle. The hæmocytozoa are mononucleated unicellular organisms and they never coalesce to form a plasmodium.

In 1890, Grassi and Felletti² proposed to divide the hæmocytozoa of man into two genera, for which they suggested the names *Hæmamæba* and *Laverania*. In 1899, Major Ross³ suggested the term *Hæmomenas* as a generic name for the parasite of sub-tertian, but this term is excluded by the name *Laverania* previously advanced. Besides, should the division into two genera be retained, I think we could not find a more suitable term for the parasite of sub-tertian than that which records the name of the great savant who discovered it.

Personally, I do not see the reason of making a special genus for the parasites of sub-tertian and quotidian on account of the shape of their gametocytes, and I think we should call the former, with Labbé, *Hæmamæba laverani*, and not *H. precoc*, as several authors have done, because the name *H. precoc* was given by Grassi and Feletti to the pigmented quotidian parasite.

Having rejected the term malaria, it becomes necessary to alter the name of the quartan parasite, which is now called *Hæmamæba malariae*, and I therefore suggest that it be called *Hæmamæba Golgi*, because it was Prof. Golgi, of Pavia, who, having made a special study of quartan fever, discovered that the different types of intermittent fever correspond to different species of parasites.

Like most unicellular organisms, no matter whether classed as Protophyta or Protozoa, the Hæmocytozoa present two different and alternating life-cycles. Considering these two cycles from the standpoint of human pathology, the one which is spent within the blood of the vertebrate intermediary host has been called the ENDOGENOUS or FEVER-PRODUCING CYCLE; the other, which is spent within the body of the arthropod definitive host, has been called the EXOGENOUS or MOSQUITO CYCLE.

I think we might with advantage adopt for the denomination of these two cycles names that would apply equally well to other organisms, and thus draw attention to the analogy which exists between the dual life-cycle of the hæmocytozoa and that of other parasites belonging to widely-sundered biological groups. The fever-producing cycle might be called the SWARMING or ASEXUAL STAGE, because during this

¹ Dr. Billet, in a recent paper on the intermittent fevers of Algeria, though still holding that tertian and sub-tertian parasites are the alternating forms of a single species, admits that the quartan parasite is a specifically distinct organism.

² "Contribuzione allo studio dei parassiti malarici." *Atti dell' Accademia Gioenia di scienze naturali in Catania*, vol. v., serie 4.

³ "Life-history of the Parasite of Malaria." *Nature*, August 3rd, 1899.

stage the parasite goes on multiplying very rapidly by an asexual method of reproduction analogous to the vegetative growth of algæ and bacteria, or to the strobilation stage of Cestoda. The mosquito cycle, or, to be more correct, the arthropod cycle, might be called the **RESTING** or **SEXUAL STAGE**, because, in this stage, the process of multiplication is a sexual one, analogous to that which produces the resting stage of Protophyta, or the resting stage of Cestoda, and because the copula or zygote becomes encysted just like the copula (resting spores) of Protophyta.

In a paper read before the Royal Society on March 6th, 1902, Prof. Ray Lankester proposed that the **SPOROZOITES**, when first introduced into the blood of man by the anopheles, should be called **EXOTOSPORES**, because, from the pathological point of view, they have been formed outside the human body. And he proposed to call **ENHÆMOSPORES** the gymnosporos, which derive from the segmentation (schizogony) of the parasite (schizont or amœbula) in the blood of the infected human being, so as to distinguish them from the spores which are inoculated by the mosquito, and derived from a sexual process of multiplication. To me, the term exotospore seems inappropriate, and, indeed, it would be absurd to call the sporozoite exotospore, within the body cavity or salivary glands of the mosquito, where we see it exclusively. The term enhæmospore is good, but to avoid an unnecessary and confusing multiplication of names, I think we should adopt the word **MEROZOITE**, which, in the Coccidiida, is applied to the analogous fission product of the schizont.

FORMATION OF GAMETES.

The formation of asexual spores (merozoites) during the swarming stage of the hæmamœbidæ is analogous to the production of zoospores in various Protophyta. The conditions favourable to asexual multiplication are those which render the vegetative growth most active and produce an abundant formation of new protoplasm. But the actual stimulus to division is a slight check to the process of assimilation and growth. In many algæ in which asexual spores (zoospores) are produced at night, the waning light and a lower temperature may be the necessary stimulus. Experimentally, a change from a strong solution of nutritive salts to fresh water will induce the formation of zoospores in plants, which would simply have gone on growing if left in the nutritive solution. In the hæmamœbidæ, the exhaustion of the nutritive supply within the enclosing corpuscle is, of course, the necessary cause of segmentation. Like many algæ which penetrate the leaves of host-plants, or like other sporozoa which live in the cells of various hosts, the hæmamœbidæ penetrate the erythrocytes simply that they may gain the advantage of a quiet, protected place for their development.

The formation of gametes in the hæmamœbidæ is likewise analogous to the formation of gametes in the Protophyta. In both the stimulus to the formation of sexual cells is given by conditions unfavourable to growth. In the common blue-mould (*Penicillium glaucum*) found on bread, fruit, &c., the *mycelium* develops special branches (*conidia*) which break up into asexual spores. But when the excessive develop-

ment of the conidia is prevented by exclusion of air, sexual organs arise on the luxuriant mycelium. The formation of gametes is an adaptive provision for the preservation of the species under unfavourable conditions, while the formation of asexual spores is simply a process of continuance and expansion. By inoculating the merozoites of the hæmamœbidæ into a succession of susceptible hosts we might continue the asexual stage indefinitely. A good illustration is that of the aphides, popularly known as plant-lice. In a favourable summer the Aphides may produce as many as twelve or fourteen generations of female insects by *parthenogenesis*. At the beginning of the cold season they produce both males and females, and the fertilised eggs of the latter can resist a very low temperature, and are known as "winter eggs." Now, if the summer aphides that multiply by parthenogenesis were not exposed to the influence of cold, but kept in a constant summer heat, and at the same time supplied with suitable food, no males would occur, and the young would be uninterruptedly produced by parthenogenesis. In fact, Réaumur succeeded in producing artificially more than fifty parthogenetic generations in the course of three or four years, all descended from one another. Probably, in the hæmamœbidæ, as in the Protophyta, only a certain number of cells in each brood become gametes; possibly the number increases as the surrounding conditions become more unfavourable. If this be so, we need not speculate on possible latent forms which have not been observed, or ascribe parthenogenesis to the gametocytes, as Prof. Grassi has done to account for relapses. Latency and relapse, in the intermittent fevers, are only clinical expressions of numerical fluctuations which occur in the successive broods of parasites in connection with conditions more or less favourable to their multiplication. In fact, we know that during latency the slightest change in the condition of the host (exposure to cold, fatigue, intercurrent disease) will give rise to a relapse.

We do not know how long the gametes can persist in the circulation; certainly, those which are not taken out of the blood by mosquitoes degenerate and disappear like Bladder-worms or the larvæ of *Filaria* if not liberated within a certain period.

A careful, continued study of segmentation (number and size of spores, disposition of chromatin, &c.) in successive broods of first uncomplicated infections of tertian fever will most probably reveal the genesis of gametocytes.

GEOGRAPHICAL DISTRIBUTION OF INTERMITTENT FEVERS.

Maps of the geographical distribution of "malaria" have often been published. In these maps, the range of the disease is usually represented by red paint uniformly brushed over all land areas between the annual isotherms of 30° N. and 70° S. The absurdity of such maps becomes at once obvious when we consider—

(1) That "malaria" is not a single disease, but a group of diseases caused by specifically distinct parasites, each one of which has its own peculiar distribution.

(2) That the intermittent fevers are strictly limited to swampy areas which are mostly found along river valleys, at the foot of mountains, and on coast districts.

When heat was considered to be the chief factor in the production of "malaria," the following law was formulated, notwithstanding all contrary evidence: "Malaria shows a progressive decrease both in extent and intensity from the Equator to the Poles, and beyond a certain limit does not occur either endemically or epidemically."¹

Now we know that heat is not a cause of the intermittent fevers, that it has nothing to do with the severity of the malignant type (sub-tertian), and that, whatever may be its influence on the epidemic prevalence of these fevers, it is certainly subordinate to many other conditions.

The recent scientific demonstration that the intermittent fevers are propagated by mosquitoes of the genus *Anopheles*, and, so far as we know, only by means of these insects, will help very much in the mapping out of fever stations, because, although the intermittent fevers may be absent in places occupied by mosquitoes known to be capable of fostering the specific parasites, they cannot extend beyond the distributional areas of these insects. Unfortunately, the geographical distribution of the various species of *Anopheles* is as yet very imperfectly known.

The available information as to the prevalence and distribution of each one of the intermittent fevers is very scanty. We know that their distribution in many places is co-extensive, and, so far as our experiments go, we know that the same species of *Anopheles* may foster the various parasites; yet there are marked differences in the seasonal prevalence and geographical distribution of each fever.

In the summer of 1899, Drs. A. van der Scheer and J. Berdenis van Berlekom² had the opportunity of investigating an epidemic of intermittent fever which broke out in Middleburg (Zealand), where for thirty years this disease had never occurred; they found that it consisted only of cases of tertian.

In the Roman Campagna, quartan, tertian, sub-tertian and quotidian are all prevalent, but each fever observes its peculiar season and maintains its usual distribution.

In Tropical Africa, sub-tertian predominates almost exclusively. Drs. Stephens and Christophers, both in Lagos and on the Gold Coast, found only the parasite of sub-tertian, and they examined the blood of 639 cases. Dr. Daniels, in Central Africa, found this parasite exclusively, in Europeans, natives and Indians. Cases of tertian fever are comparatively rare on the West Coast. On the East Coast, Prof. Koch found the *Hæmaphysa vivax* in 10 per cent. of all cases of fever. Quartan fever seems likewise very rare. Major Ross mentions it as occurring in Sierra Leone.

I am sure that a careful comparative study of the distribution and ecology of these fevers by competent observers would elucidate many obscure and important points in the life history of their respective parasites. Quartan fever is, perhaps, the easiest to investigate. It has a wide distribution, but its

stations, or endemic centres, are very much scattered and strictly limited to certain localities which offer special conditions as yet undetermined. A classical example is that recorded by Troussseau many years ago. "Tours and Saumur, which are both situated upon the left bank of the Loire, would seem to be under the same climatic and telluric influences, yet in Tours we find only the tertian variety, while all the quartan cases which I have seen were in persons coming from Saumur, from Rochefort, or from some other region. One of the facts which made the deepest impression upon me was the following: Fourteen soldiers from Saumur came to Tours to testify at a court-martial; they had scarcely been ten days in the latter city when nine of them were obliged to go to the hospital suffering from quartan fever, the germs of which they had certainly contracted in Saumur, since all the fevers observed in Tours and its environs were of the tertian type."

While carrying out Dr. Manson's experiment in the Roman Campagna in 1900, Dr. G. C. Low and myself,³ examined every case of fever that occurred in the district of Ostia, but we found no cases of quartan, whilst at a few miles distance, near the convent of the Tre Fontane, there occurred several cases. In connection with this fact, I may mention that although we hunted daily for mosquitoes, we did not find one single specimen of *Anopheles bifurcatus* in the district of Ostia, but, at the Tre Fontane Prof. Bastianelli found several specimens of *Anopheles bifurcatus*, together with specimens of *A. maculipennis*, which were everywhere exceedingly abundant. Whether the species of *Anopheles* has anything to do with the peculiar distribution of quartan fever I am not prepared to say, but Profs. Bastianelli⁴ and Bignami state that whereas the experimental infection of *A. maculipennis* with tertian and sub-tertian parasites is comparatively easy, that with quartan parasites is very rarely obtained. They believe that this is on account of the scarcity of gametes in the blood of quartan patients, rather than to the insusceptibility of *A. maculipennis* for this particular parasite.

It is to be hoped that we shall soon possess records of careful experiments on the infection of many species of *Anopheles* and other mosquitoes with the parasites of the various intermittent fevers.

DERIVATION OF THE HÆMOCYTOZOA OF MAN.

So far as we know, the parasites of quartan, tertian and sub-tertian are found in man only. Probably the reason of this limitation is that they have become specialised, like *Tania saginata*, *Pediculus capitis*, *Phthirus inguinalis*, and other parasites of man belonging to very different biological groups.

In recent years the blood of numerous animals has been examined for hæmocytozoa, and we have learnt that these parasites have a wide zoological distribution, but in no other vertebrate have been found the hæmocytozoa peculiar to man.

Several kinds of Hæmocytozoa have been found, and zoologists have grouped them into three distinct

¹ Hirsch's "Handbook of Geographical and Historical Pathology."

² *British Medical Journal*, January 26th, 1901.

³ In this we were very kindly and ably assisted by Dr. Bartera of the Italian Red Cross Ambulance.

⁴ "La Malaria e le Zanzare." Comunicazione fatta al X^o Congresso della "Società Italiana di Medicina interna." Roma, 1899.

genera, called respectively: *Hæmamaeba*, *Piroplasma*, and *Hæmogregarina*.

The genus *Hæmamaeba* is found in man, in bats, in monkeys, and in various kinds of birds during its asexual stage, and in no other class, unless we accept a still doubtful case in a tortoise (*Trionyx indicus*). It is fostered, as far as we know, by mosquitoes during its sexual stage.

The genus *Piroplasma* is peculiar to dogs, horses, sheep and oxen during its asexual stage; the species parasitic in oxen and dogs are transmitted by ticks from one intermediate host to another. Of the others we have no knowledge.

The genus *Hæmogregarina* contains a large number of species and is possibly the oldest. It is essentially parasitic in amphibians and reptiles. We know nothing as to the way in which it is propagated.

Like man, so several animals harbour more than one species of hæmocytozoa. Thus, in the pigeon (*Columba livia*) we find both *Hæmamaeba Danilewskyi* and *H. relicta*; in the edible frog (*Rana esculenta*) we find *Hæmogregarina ranarum*, *H. splendens*, and *H. magna*; and in oxen we find two kinds of *Piroplasma*, if I am right in surmising that the small parasite is a distinct species, and not a mere form of the large *Piroplasma bigeminum*.

The majority of hæmocytozoa, like those of man, are confined to one particular host, but some have two or more hosts. Dr. Low and myself found that *Hæmamaeba melaniphæra*, a parasite discovered by Dr. Dionisi in the mouse-coloured bat (*Myotis-myotis*), is also found in *Myotis capaccinii*. *Hæmogregarina Lacazei* is found in *Lacerta agilis* and in *L. muralis*. *Hæmogregarina lacertarum* is found in various kinds of lizards, and both *Hæmamaeba Danilewskyi* and *H. relicta* are found in many birds of different genera.

Thus we see that the Hæmocytozoa conform to the general laws of parasitism, and that by examining their life-history comparatively with that of other better-known parasites, we may possibly succeed in discovering their derivation.

The hæmocytozoa, like all other parasites, must have originated from non-parasitic forms, but the species now found in man and other vertebrates probably derived from earlier species already parasitic on other hosts. Modern research has shown that species split up into different varieties or species, according to the particular host they inhabit, and that they often become so greatly modified and changed, in order to adapt themselves to certain hosts and to certain conditions, that they become absolutely dependent upon these hosts, and the presence of these conditions for their existence. Such changes are actually taking place, and with certain organisms we can induce them experimentally. In fact, bacteria that have been fed for several generations on media rich in proteids refuse to grow on media rich in carbohydrates. The Stem Eel-worm (*Tylenchus devastatrix*) lives and reproduces in various cultivated plants, such as rye, oats, stored onions, hyacinths, buck-wheat, potatoes, and clover, and in wild plants,

such as *Poa annua*, *Anthoxanthum odoratum*, *Dipsacus silvestris*, and *Polygonum persicaria*, but not to the same extent in all. However, Eel-worms, of which the progenitors have developed for many years exclusively in rye and buck-wheat, are not easily transferred to another kind of plant, or at any rate, they do not multiply vigorously there.

Hæmamaeba Danilewskyi is found in a large number of birds; in the course of evolution it will probably split up into as many varieties or species as there are hosts, just as Rust-fungi have split up into different varieties according to their particular host-plants.

When we find in the same host several parasites specifically distinct, but belonging to the same genus and living in exactly the same conditions, it is not unreasonable to surmise that, in the first instance, they probably derived from other hosts in which they acquired the peculiar morphological and biological characteristics which distinguish them now in the new host.

Clinically and pathologically, blackwater fever is a disease exactly similar to the hæmoglobinuric fever of cattle. Should the parasite of blackwater fever prove to be an exceedingly minute species of *Piroplasma*, we might surmise that the disease had been acquired in the first instance from that group of animals in which the genus *Piroplasma* thrives. This hypothesis concerning the etiology of blackwater fever is based on the following facts:—

(1) That our inability to discover the specific parasites of certain diseases, is possibly on account of their diminutive size. In fact, some known parasites, such as the microbe of cattle peri-pneumonia, are so exceedingly small that we are unable to see them with our highest powers, unless they be aggregated into masses.

(2) That while *Piroplasma canis* and *P. bigeminum* are comparatively large parasites, *P. ovis* is very minute. Indeed, when first discovered by Babes, it was described as a micrococcus and called *Hæmatococcus*.

The hæmocytozoa of man so far described belong to the genus *Hæmamaeba* which is restricted to mammals and birds. Now, considering that, as a rule, we become infested with the parasites of those animals with which we come most frequently in contact, it would not be unreasonable to suppose that we may have derived the parasites of our intermittent fevers from those animals, like certain birds and bats, which nest in cities under our very roofs, and that the parasites have been transmitted from these animals to man by those mosquitoes which have also linked themselves with man after the fashion of the cockroach and the house-fly. However, all attempts to infect various species of animals with the hæmocytozoa of man by injecting blood containing these parasites have been uniformly unsuccessful. The few experiments made by means of infected mosquitoes have likewise proved negative. But experiments of the kind have been as yet too scanty to allow of any definite statement. Prof. Dionisi¹ inoculated four men with the blood of bats containing their peculiar hæmocytozoa, and his experiments were inconclusive.²

¹ Report on Two Experiments on the Mosquito-Malaria Theory. *Medico-Chirurgical Transactions*, vol. lxxxiv. London, 1902.

² "La malaria di alcune specie di Pipistrelli." *Annali d'Igiene sperimentale*. Roma, 1891.

³ One man inoculated with blood of the mouse-coloured bat (*Myotis-Myotis*), containing a hæmamaeba somewhat similar to

But the mosquitoes which propagate the parasites of the intermittent fevers do not feed exclusively on the blood of vertebrates, and it would be rash, therefore, to venture any opinion as to the derivation of the hæmocytozoa of man until we have a definite knowledge of the feeding range of the Anophelina. The general opinion is that mosquitoes in the perfect state are essentially phytophagous. Several entomologists have seen them settled on plants sucking the juices of flowers, and Lieut.-Col. Giles,¹ who examined the contents of the stomach of mosquitoes in India, states that "the most usual recognisable constituent in both sexes was pollen." In captivity they will readily imbibe the juices of fruit.

Mr. L. O. Howard² says: "The female mosquitoes are normally without much doubt plant-feeders. Why they should draw blood at all is a question which has not been solved. It has been surmised that a supply of highly nutritive fluid is necessary for the formation of the eggs, but this supposition is at once emphatically negated by the fact that mosquitoes abound in regions into which warm-blooded animals never penetrate. The statement which the writer has elsewhere made, that not one in a million ever gets the opportunity to taste the blood of a warm-blooded animal, is unquestionably an under-estimate. There are in this country enormous tracts of marshy land into which warm-blooded animals never find their way and in which mosquitoes are breeding in countless numbers, and when we get within the Arctic Circle and other uninhabited regions the point is emphasised." Some species of mosquitoes may be strictly phytophagous, but the Anophelina are decidedly carnivorous. The fact that female mosquitoes can be kept alive for months on the juices of fruit does not prove that the sap of plants is their natural food. On the contrary, however paradoxical the proposition may seem, it proves that their life has been prolonged by the want of suitable food. Indeed, it is a well-known fact that the female mosquito dies after having exhausted her ovaries by successive ovulations, but that it may live for months, exhibiting that remarkable endurance to want of food which is common to many insects, so long as unfavourable conditions prevent oviposition. Mosquitoes are exceedingly voracious during their larval stage, because that is a period of rapid growth and demands a large amount of food. But the insect in the perfect state is, as a rule, short-lived, does not grow, and requires very little food, all it needs is a certain amount of water, especially in very dry weather, and this it imbibes from plants. The male anopheles can only sip water, his mouth-parts, like those of the males of other Diptera (*Simu-*

the tertian parasite, had a paroxysm of fever after twenty-eight days.

Another man inoculated with the blood of *Miniopterus schreibersii* containing a parasite resembling that of quartan fever suffered after fifteen days from two paroxysms of fever of tertian type.

Of two men inoculated with the blood of the noctule *Vesperugo noctula* containing an unpigmented parasite, one, after twelve days, suffered from fever of a sub-tertian type, the other remained immune. In none of these cases were parasites ever found although the blood was examined in each case for over a month.

¹ "A Handbook of the Gnats or Mosquitoes." London, 1902.

² "Notes on the Mosquitoes of the United States." Bull. xxv., New Series, U.S. Dept. Agriculture, Washington, 1899.

lida, *Tabanida*), are inefficiently developed for any other purpose. But the female mosquito needs a certain amount of food-reserve for her ova, and this requirement is well exemplified in *Rhipicephalus annulatus* and other ticks, the females of which, after fertilisation, suddenly gorge themselves enormously with the blood of the host on which they have lived parsimoniously throughout the various stages of growth and metamorphosis.

Mr. Howard's statement that not one in a million mosquitoes ever gets the opportunity of biting warm-blooded animals within the Arctic Circle is, I think, not under-estimated, but greatly exaggerated. Certainly the Mammoth and the Bison roam no longer over the vast Tundras, but in their short, hot summer, these regions become richly populated with Arctic mammals and migratory birds.

But the blood of mammals and birds is probably not essential; that of amphibians, of fish and of insects may serve the same purpose, and, in fact, Mr. J. Turner Brakeley³ saw a fresh-water tortoise surrounded by mosquitoes; Mr. C. H. Murray saw the mosquitoes alight upon the heads of young trout as the latter came to the surface of the water; Dr. H. A. Hagen caught a mosquito which was feeding upon the chrysalis of a butterfly; Mr. Theobald⁴ saw *Culex migratulus* sucking the body of *Chironomus* and other small diptera; and Dr. H. A. Veazie saw mosquitoes stinging the Cicada and its pupa.

The fact that mosquitoes suck the blood of other insects is very important, because it shows that we may have to search a wider field for the derivation of our hæmocytozoa. The sporozoa are widely distributed amongst the arthropoda: I need only mention the silkworm parasite (*Glugea bombycis*), which has cost communities millions of pounds.

It is not unreasonable to suppose that vertebrates and invertebrates may both foster the asexual stage of the hæmocytozoa. We have many examples to prove that both the intermediary and the definitive host of parasites may be represented by animals belonging to widely-sundered groups of the animal kingdom, while closely-related species are not susceptible. Should invertebrates be capable of fostering the asexual stage of the hæmocytozoa which are known to live in the blood of vertebrates, we might be able to explain many obscure points in the geographical distribution and epidemiology of the intermittent fevers. Already in another paper on the "Principles Determining the Geographical Distribution of Disease,"⁵ I suggested that the disappearance of the intermittent fevers from England and other places, notwithstanding the abundance of suitable mosquitoes and other favourable conditions, might be possibly connected with the extinction of some animal, or perhaps some plant, which may have been a necessary link in the chain of their natural history, and I mentioned the *Large Copper Butterfly* as an example of a species which has lately become extinct within the ague districts of England.⁶

³ Howard, *loc. cit.*

⁴ "Monograph of the Culicidae or Mosquitoes." London, 1901.

⁵ JOURNAL OF TROPICAL MEDICINE, April 1st, 1902.

⁶ Mr. Theobald believes that the disappearance of ague from England may have been due to the fact that *Anopheles maculipennis* has lost the habit of blood-sucking in this country. He

From what I have said, it is obvious that our information is as yet too scanty to allow of any serious conjecture as to the derivation of the parasites of the intermittent fevers.

PROPHYLAXIS.

With regard to prophylaxis, I wish to say a few words on a method of prevention which I think has not yet received adequate consideration. I mean the study of the natural enemies of the malaria-bearing mosquitoes. In the domain of agriculture we have already achieved wonderful success by pitting against certain injurious insect-pests their natural enemies and parasites. A good example is that of a fungus *Tsaria destructor*, which Prof. Metzhinoff employed to destroy the larva of the Beet-chaffer (*Anisoplia agricola*), which caused much damage to the beet-fields in Russia. The use of the fungus, combined with a suitable rotation of crops, answered most admirably, and, indeed, after a few years the beet crop became so great that it was thought advisable to let the beetle continue its ravages and thus become once more the regulator of the produce! Surely, it is reasonable to expect similar results in combating those insects which foster and disseminate the pathogenic parasites of man. Already a number of predaceous and parasitic enemies of the Anophelina have been described, some of them exceedingly injurious to the genus and attacking it either in the larval stage, or in the adult stage, or again in both; but the information so far collected is still very scanty and no experiments have been made to test its practical value. In connection with this, I wish to draw attention to the fact that very frequently the intentional or accidental introduction of certain species has caused the disappearance of allied native species, not because the introduced species preyed upon the native one, but on account of that intense rivalry which takes place universally amongst closely-related species which use the same food, the same hosts, and the same breeding grounds; or, sometimes, on account of parasites tolerated by the introduced species, to which they have become inoffensive, but deadly to the native ones. Thus the advance of the Colorado beetle (*Leptinotarsa decem-lineata*), and its prodigious increase on the cultivated potato, has caused the disappearance of the native species (*Leptinotarsa juncea*) from Illinois, Delaware, Maryland, and New Jersey, where it was formerly very abundant. Again, *Pieris rapa* has replaced, in America, the native *Pontia oleracea*. Walsh pointed out, thirty years ago, that the westward spread of the European *Mytilaspis pomorum* caused the gradual local disappearance of the native *Chionaspis furfur*. Hubbard has shown that the increase of the imported *Mytilaspis citricola* in Florida was followed by the decrease of *Mytilaspis gloverii*, which, though not native, was an earlier importation. A well-known example is that of the Brown rat (*Mus decumanus*) from East Central Asia, which has practically expelled the Black rat (*Mus rattus*) from Europe, just as the latter has been ejecting weaker

rodents from South America. Many other instances might be adduced, and in some cases the biological effects of the introduction of an alien species are very considerable. The introduction of goats into St. Helena caused the destruction of a whole flora of forest trees, and with it the extermination of all the insects, mollusks, and perhaps birds dependent upon it.

The chief preventive measure against malaria is, undoubtedly, the drainage and cultivation of the swamps and meres which are the breeding grounds of the malaria-bearing mosquitoes; and such measures have always proved efficient whenever they have been thoroughly put into act and fact, from the oldest times when Empedocles reclaimed the fever swamps of Agrigentum. But when, owing to economical or other reasons, the obliteration of the swamp is not possible, we must necessarily have recourse to other means. The kerosene treatment of the anopheles' breeding-grounds is practically impossible over wide areas of uncultivated land. The weeding of small water-courses by men or cattle, as practised in the Pontine Marshes, is of little importance. The routine administration of quinine to the malaria-bearing native population in order to avoid the infection of new broods of mosquitoes is, in most regions, beyond possibility. But a combination of these methods, and the general use of mosquito netting are bound to yield good results. I have no doubt, however, that sooner or later we shall find some plant that will prevent the breeding of mosquitoes, or some innocuous gnat which will displace the dangerous anopheles, or some other animal which will prey upon it and prove as successful as the ladybird (*Vedalia cardinalis*) with which Dr. Riley saved the great orange industry of California that was being destroyed by a scale insect, the *Icerya purchasi*. Already we know that the common duckweed (*Lemna*) will prevent the breeding of anopheles in small quiet pools by completely covering the surface of the water, and we know that whenever several species of mosquito larvæ are found together in the same body of water, one of them is usually greatly preponderant, the others being apparently ousted by the most successful. Indeed, the larvæ of *Corethra* and *Psorophora* prey on other larvæ of *Culicidæ*. We know also that the larvæ and pupæ of mosquitoes are devoured in great numbers by water-fowl, small fish, or by the predacious larvæ of many aquatic insects (*Dytiscidæ*, *Hydrophilidæ*, *Gyrinidæ*, *Notonectidæ*, *Odonata*), and that they are often destroyed by parasitic algæ. Likewise, the swarming adult insects may be killed off by bats, nighthawks, swallows, martins, flycatchers, lizards, and dragon flies, or they may be attacked by ecto-parasitic acari, such as *Acarus culicis*, or by internal animal parasites, such as *Filaria*, *Trematodes* and *Sporozoa*; or, again, by certain fungi, such as *Entomophthora culicis*, which at times causes veritable epidemics amongst them.

I am fully aware that certain experiments with natural enemies have utterly failed, but probably, in such cases, the life-history of the organism employed was imperfectly known, and consequently some necessary intermediate host, some associate-organism, or some special condition of environment was neglected.

says: "I have never known this species to attack human beings, and yet I have lived in districts where it often occurs in great numbers and other mosquitoes are troublesome."

We cannot always unravel the tangled skein of inter-relationships which make up the peculiar ecology of each organism.

In a few instances, the intentional introduction or cultivation of natural enemies have proved positively disastrous. Thus the sparrow introduced into the United States to destroy canker-worms has become a veritable pest throughout all the Austral and Transition regions of North America, while the place of the injurious insect it was imported to destroy has been taken by another and worse insect pest which it will not touch. Another instance in point is that of the common mongoose of India (*Herpestes mungo*), which was introduced into Jamaica, in 1872, for the purpose of ridding cane-fields of rats. The rats were soon annihilated, but the evil results of the introduction of the mongoose far outweighed the benefits rendered to the sugar plantations.

Should we fail to destroy mosquitoes by means of their natural enemies, there would still be the hope of finding some hyper-parasite that might prevent the development of the parasites of intermittent fever within the body of mosquitoes. Indeed, the disappearance of the hæmocytozoal fevers in places in which the suitable mosquitoes abound may possibly have been brought about by the agency of hyper-parasites. The "Blackspores" or "Brown bodies" discovered by Major Ross and believed at first to represent a special stage in the life-history of the hæmocytozoa are probably hyper-parasites.

A well-known instance of hyper-parasitism is that of Ichneumon flies. The parasitic larvæ of these flies exercise a constant check upon the increase of aphides and other insects injurious to plants, but they are likewise themselves kept within due bounds by hyper-parasites, which detect at once a "stung" aphide or caterpillar, and deposit their eggs within the body of the internal parasite. The larvæ hatched out of these eggs may also be attacked by similar foes, whose offspring in turn may suffer in like manner. It is a wonderful process carried on indefinitely, far beyond our powers of investigation, and of which those "nested" Japanese boxes, which are enclosed one within another, are an apt illustration.

THE AFTER-COMPLICATIONS IN THE OPERATIVE TREATMENT OF CATARACT.

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In the following paper it is proposed to take up in turn some of the complications which one meets with in the treatment of cataract after operation.

These suggestions must be read in connection with my previous paper, which appeared in the JOURNAL OF TROPICAL MEDICINE for September 1st, p. 263.

While writing, I have under review (1) about 1,200 cases which, in my early days, I operated on as far as possible by the Simple Operation and by the ordinary methods; and (2) another 800 cases in which the Combined Operation was always performed if possible, and in which MacKeown's valuable method of irrigation of the anterior chamber was used as the routine procedure.

Each heading may now be taken up in turn.

(1) *Want of sound union of the section* may almost always be set down to some defect in the nutrition of the patient. Most of the patients are advanced in years (*i.e.*, above the average age of those operated on). In one series of 77 cases, the section leaked in 7, or in 9.09 per cent. At one time in this series, several ununited sections occurred together; this led me to investigate the cause, and I found that the cook-contractor was robbing the patients' food. Attention to this point led to an *immediate* healing of all the sections.

In two other series, comprising 750 cases, delay in union was met with in 66, or in 8.8 per cent. The limits of delay were from two to sixteen days, most of the cases healing on the fifth or sixth day; this was probably due to the routine commencement of treatment on the fourth or fifth day. If the section has not united by the fourth day, two drops of solution of arg. nit. (gr. 3 ad. oz. 1) are instilled on to the section every morning; the bandage is continued over both eyes till the chamber has been closed for at least two days.

I have been accustomed to think very lightly of this complication, as my own experience had closely coincided with that of others, as to its seldom leading to trouble. Recently, however, I lost an eye from sup-puration setting in on the fourth day of an open section; there had been slight conjunctival congestion before operation. This misfortune has invested "leaking section" with an increased importance in my eyes, especially if there has been any doubt about the state of the conjunctiva beforehand.

When there is reason to doubt the patient's powers of repair, it is well to take up a small conjunctival flap with one's section. If the section advocated in my previous paper be used, this can always be easily done, even although, as there pointed out, the wound lies almost entirely in the corneal tissue, for situate as it is in the limbus, it only encroaches on the sclerotic in its external scleral process, and yet it gives us ready access to the conjunctival margin.

(2) *Injury to the eye during healing.*—This is generally due to the patient finding the eye itchy and rubbing it. It practically always takes one of two forms, either the section is burst, or the chamber fills with blood.

In 827 cases I find that the *patient burst his section* eighteen times, or in 2.17 per cent. This accident usually occurs within the first week, but may rarely be met with later. It is commonly unattended with symptoms, but in two cases it was accompanied by severe pain and obstinate vomiting. Some hæmorrhage into the chamber not uncommonly attends rupture of the section, and a subsequent capsulotomy is therefore sometimes called for. I can find no case in my notes in which this accident led to loss of vision, nor can I even find that it markedly unfavourably influenced the results of the operations.

In the same 827 cases, *hyphæma* was found in 35, or in 4.24 per cent. In one case it was due to a fit of coughing, in another to a blow (the eye striking the bed-post), and in most of the rest to the patient rubbing the eyeball. This accident may occur as early as the fifth day, but is more often met with later. Speak-

ing generally, an injury to the eye during the first week will burst the section (with or without some hæmorrhage); whereas an injury at a later date leads to *hyphæma*, the section being by then fairly strongly healed.

Here, again; I cannot find that any eye has been lost, or that vision has been sensibly lowered by the accident; but it is my impression, that if one were able to follow these cases for some months, many of them would benefit by a capsulotomy. As a rule, it takes several days for the absorption of blood from a chamber, if the hæmorrhage has been free; I have, however, seen one case in which a chamber full of blood cleared completely in twenty-four hours.

Bleeding into the anterior chamber, which occurs after the chamber has once cleared of blood after operation, may almost certainly be attributed to an injury, and usually in the East to the patient having rubbed the eye.

Hæmorrhage occurring at the time of operation may come from the iris or from the conjunctiva. I have never seen reason to think it may come, as has been stated, from the sclerotic. With MacKeown's irrigator, the blood can be easily and safely washed away; and in the last 750 cases I have operated on, I cannot remember, or find notes of, one in which the chamber was blood-stained the morning after operation. Need one point out that this lessens the strain put upon the absorptive powers of the eye, and so makes for safety?

(3) *Faulty apposition of the flap* is discovered at the first dressing. It is more likely to occur in troublesome patients, who will not allow the toilette of the wound to be carefully attended to, and especially in those whose vitreous has escaped or threatened. The immediate agent in producing the complication is the edge of the upper lid, which catches in the free edge of the flap, and either pushes it downwards, or even turns it back on itself. Want of elasticity of the cornea predisposes the eye to this accident. When one sees that the section-flap is inclined to turn backwards or to slip down, the lid should be lifted off the globe either with a finger and thumb, or, if necessary, with a pair of forceps, and held thus while the flap is reposed in position, the patient being directed to look downward for the purpose; he should then be told to look upwards. By the latter movement he carries the section well under cover of the lid, which can be gently lowered on to it, and the eye is at once closed and bandaged.

If, in spite of care, the flap has set in bad position, it is better to instil a drop of cocaine, and repose it with a spatula at the first dressing.

In 827 cases I find that the section was in faulty apposition in 9, or in 1.08 per cent.; it was satisfactorily replaced under cocaine in 6, all obtaining good vision; of the remaining 3, one obtained very indifferent sight, the other two recovering with good vision. It is obvious, however, that the defective apposition must have led to increased astigmatism.

(4) *Cystoid cicatrix* is an uncommon condition in my experience, presumably because the irrigator effectually washes any capsule tags into the chamber, and therefore out of the section. In 827 cases I met with it once, and as pressure and bandaging failed to relieve

the condition, I freely excised the bulging section, with the result that sound union was at once obtained.

One may meet with a bulging section in a condition apart from that just described; here it is not a portion of the section that yields, but its whole extent, and further it is not the section itself which is to blame, but the tension of the eye, which is raised by some post-operative condition. In the only case of this nature I have met with in over 2,000 operations, the tension was normal before operation, the chamber was of normal depth, the cataract cortico-nuclear, with thickening of the capsule. The patient, a male, aged sixty years, had been treated before operation for slight catarrh of the conjunctiva; his eye had been useless for two years, and he could only detect hand movements with it. The lens was removed by the combined operation, and a large amount of doughy cortex was washed out with MacKeown's irrigator; a piece of thickened capsule was removed with iris forceps. The section healed, and the eye looked well on the third day; on the fifth day the section began to bulge, and the bulging, which was apparently due to over-distention of the vitreous, increased in spite of firmly-applied pressure; on the fifteenth day the conjunctival membrane, which apparently formed the whole covering of the section, was carefully snipped away under cocaine, and the projecting vitreous was snipped off with scissors; the section then healed, and on the twenty-sixth day the patient presented a firm, quiet eye. It seems probable that in this case there was some obstacle to the outflow of fluids from the vitreous leading to an elevation of the tension of the globe. The patient recovered with excellent vision. Possibly this is a case of very early post-operative glaucoma.

(5) *Striped keratitis* is as common as it is unimportant. Whether it is due to wrinkling of the posterior corneal layers, or to damage to the posterior lining membrane and endothelium, one thing at least is certain, viz., that the delivery of a large, hard lens under any circumstances, or the delivery of any lens through an insufficient section, is likely to be followed by striped keratitis. It is seldom that this transient condition calls for any treatment, but if the resolution of the opacity is delayed, it is well to apply a yellow oxide ointment to the conjunctival sac. It is important to distinguish a quite different class of cases, viz., those in which *true keratitis* occurs.

(6) Such a misfortune is to be apprehended after operation on a cornea which has at some previous time been ulcerated, and it is essential to examine the cornea beforehand in every case, and to abstain from operation if the mischief be still active. Even when the eye is quiet at the time of inspection, I would counsel delay for several months if the trouble has been recent. Some years ago I met with a very distressing case in which troublesome ulceration of the cornea followed a too free use of cocaine, and this has led me to be very sparing of cocaine in any case in which there seems reason to doubt the absolute healthiness of the cornea.

(7) A certain amount of *catarrh of the conjunctiva* is not uncommon after extraction. When it calls for treatment, this should be *mild*, and on ordinary lines.

The same remarks apply to congestion of a pterygium complicating the after-course of our cases. Such an

accident will be rare if the pterygium is avoided by the knife in section.

(8) *Spasmodic entropion* is an uncommon complication met with in old people. It occurred 5 times in 827 cases, or in 0.60 per cent. It is readily remedied by releasing the eye, and if this be promptly done, no serious consequences follow; any delay in so doing will lead to corneal complications of a very serious character. Of the 5 cases, 4 obtained good and 1 poor vision.

(9) The question of *Iris-prolapse* has been already dealt with under that of the advantages of the combined operation in my previous paper. If iridectomy be performed, and the iris edges be well replaced by the aid of MacKeown's irrigator, it will be but seldom that symptoms will arise demanding the reopening of the section and the removal of the impacted portions. In 250 cases, I had to resort to this measure only twice, or in 0.8 per cent. In 4 other cases tags of iris were impacted, but they were very small and did not give rise to any marked symptoms.

When signs of ciliary congestion or of iritis are present in consequence of this complication I hold strongly that it is mere tinkering to "abscise" the prolapsed iris; the section should be freely reopened along the whole extent of the prolapse, and the latter should be liberated from the abnormal attachments, and freely excised, the iris edges being then carefully replaced in the chamber. The operation is a difficult one and requires dexterity, but when well done it gives the patient his best chance of good and *permanent* vision. It is essential to perform it *as soon after the occurrence of prolapse as possible* for two reasons: (1) that the iris is not given time to form dense adhesions, and can therefore be readily detached; and (2) that cocaine acts much better on uninflamed structures than on those which have been for some time inflamed. Chloroform is objectionable, as the efforts to pull the eye down with the forceps menace the integrity of the vitreous.

I find that, in 827 cases of extraction, the above operation was performed 10 times for prolapse after 69 simple operations, with good results as to vision in all; it was performed 14 times for prolapse of the iris edges after 758 combined operations, with 13 good results and 1 poor one. The larger percentage of prolapse amongst the combined cases is largely due to the fact that, except in a first series of 77, the simple operation was only performed when an iridectomy could not be performed on account of the patient being refractory, and consequently the risk of prolapse was then greater.

(10) Septic post-operative conditions are amongst the gravest we have to do with. As soon as signs of acute iritis, iridocyclitis, or keratitis show themselves, I order the patient the time-honoured blue pill and black draught in a dose proportioned to his strength, four leeches are applied to his forehead and temple, and their effect is exaggerated by subsequent fomentations three or four times daily for the next few days; the conjunctival sac is kept immersed in warm chinosol solution (1/3,000) for ten minutes at a time immediately after each fomentation; the method employed is the excellent one recommended by Professor MacKeown; with the patient on his back, the head on a low pillow,

the chin well raised, and the face turned away from the side of the affected eye, one takes advantage of the cup formed by the nose on the inner side and the projecting margins of the orbit above and below to create a natural basin of antiseptic fluid in which the open eye can freely soak.

Atropine and cocaine are freely exhibited, every care being taken to avoid atropism by closing the lachrymal canaliculi with finger pressure for a minute after each instillation.

When the acute symptoms are disappearing under this treatment, blisters are applied to the temple if there is any undue delay in convalescence, and at a later stage still I do not hesitate to place a seton in the temple. I am satisfied that both blisters and setons at this stage are powerful and valuable aids in the treatment, notwithstanding the decided opinions one reads of to the contrary.

From the commencement the patient is put on bark and ammonia, and a light nutritious diet is given, with the addition of stimulants. The improvement in a number of the cases after the exhibition of stimulants has sufficed to prove their value to me, especially in the later stages of the inflammation.

The above method of treatment has given me most satisfactory results on very many occasions when the eye seemed at first to be very gravely threatened. As an instance one may cite the case of a high-caste and very wealthy native patient who refused to have any bandage on that was not made of silk (for caste reasons); the silk creased, folded and slipped off, and then I have no doubt he rubbed his eyes; at my next visit he was suffering from an extremely acute iritis, and the case looked almost hopeless; under treatment he made such a good recovery that he could without difficulty read the smallest newspaper print, and he was thus able to resume an active part in his very extensive business as a native banker.

On the other hand, there are many cases which steadily go to the bad in spite of any treatment. I have tried reopening the section and freely irrigating the chamber with chinosol solution, and again with sterile normal saline solution; the antiseptic seemed to modify and check the intensity of the inflammation in the case it was tried in, but that is the most one can claim for it. My own belief is that inflammations of a septic type which show themselves within twenty-four hours of operation are in more than 50 per cent. of the cases hopeless from the first, be the treatment what it may. The later the inflammation appears, the less cause is there to fear for the safety of the eye, and the more cause to look for some factor in the state of the patient's general health.

Among the septic complications in my notes of 750 cases, there were 3 cases of panophthalmitis. It is my custom to enucleate as soon as I am satisfied that suppuration of the globe has definitely declared itself. Amongst my first 1,100 cases I have notes of one in which for a reason I have since forgotten I delayed enucleation for several days. Finally, the ball was removed; the same night hyperpyrexia came on, and the patient died within sixteen hours of operation. I unfortunately did not see him, and a *post-mortem* was refused. My treatment in enucleating the eye was adversely criticised, but I blame myself not for operat-

ing, but for delaying the operation. Others may consider that I should have eviscerated. In the 1,100 cases amongst which this occurred, panophthalmitis had appeared 11 times.

(11) *Secondary Glaucoma* appeared in 3 cases out of my first 1,200. With the doubtful exception of one case already commented on in this paper, I have not met this complication in my last 800 cases, and I attribute this immunity to the thorough way in which MacKeown's irrigator enables one to complete the toilette of the eye, and thus to guard against the impaction of loose ends of capsule in the section.

It is likewise to the irrigator that I have to ascribe my freedom from bubbles of air left in the eye after operation, a complication that figured twice in my first 1,200 cases, but never since.

(12) The question of the best method of dealing with cases of *Secondary Cataract* requires for its solution a clear preliminary understanding of what we mean by the term in question. It would not be wide of the mark to state that the expression is so loosely used as to include any obstacle to clear vision after operation which has its seat between the plane of the iris and that of the anterior of the boundary layer of the vitreous, both the structures named being included.

We can classify these secondary membranes on a basis which is at once morphological and surgical. Thus we meet with the following:—

(1) *Thickenings of one layer of the capsule, practically always the anterior, which existed before the extraction.* These should, and easily can, be dealt with at the operation, either by removing a large central portion of the capsule with capsule-forceps before delivering the lens, or by inserting a pair of iris-forceps after the lens has escaped, and so extracting the membrane. The latter is my own practice, but it is a manœuvre which I would not recommend to any surgeon who is not confident of his powers of operating, as a false move on the operator's part, or a sudden movement of the patient's, will gravely menace the vitreous. A quick, light touch is essential for safety, but when the manœuvre is successfully carried out the result is all that can be desired. I find that in 750 consecutive operations portions of capsules were removed in this way 36 times, or in 4.8 per cent. In 5 the vitreous either threatened or actually escaped in small quantity, in 5 the healing of the section was delayed for some days, and 1 suffered from a severe attack of keratitis. In 33 cases the visual result was good, and in 3 it was poor.

It is by no means easy to always diagnose slight but important thickenings of the anterior capsule before the escape of an opaque lens. When this diagnosis can be made, it will be well to use capsule-forceps, and so ensure the removal of the opacity before the lens escapes.

(2) *The inclusion of cortical matter or of unabsorbed blood between the two layers of the capsule.* This condition is probably due to the adhesion of the edges of the capsule wound to the posterior layer, a condition which results in the shutting off of the contents of the capsule from the aqueous fluid, and so effectually stops absorption. A proliferation of the cells lining the capsule is, in many cases, doubtless, an added factor.

MacKeown's method of washing out the capsule greatly diminishes the risk of occurrence of this kind of opacity, as it clears the capsule of blood and of lens debris in a most effectual manner. In cases of post-operative hyphæma a secondary opacity of this nature is very liable to form.

(3) *Crinkling of the posterior capsule.* This comes on gradually some little time (weeks, or even months) after operation, and sometimes most unfairly discredits the original operator in the eyes of the patient. It is not impossible that the anterior limiting layer of the vitreous sometimes takes a part in this change.

(4) *Eccentric position of the aperture in the capsule* is quoted by some authorities as a cause of secondary cataract. It is a condition of which I cannot speak from experience.

For the conditions described under headings 2 and 3 (and I have no doubt 4 may be included), there is no method to beat dissection with two needles. It has been my good fortune never to have met with an accident after this operation, and in the course of the after-treatment of 2,000 and more cases I have performed it frequently. Possibly this satisfactory result is largely to be ascribed to the fact that it has been my custom to look upon this little operation as one calling invariably for the same strict observance of antiseptic treatment as an operation for primary cataract. There is a considerable difference of opinion amongst surgeons as to the best period of the case for this procedure: some have advocated a very early interference on the ground that the capsule is then more easily torn, whilst others have laid stress on the advisability of a long delay before submitting the eye to a second operation.

There is no doubt in my mind that to submit the eye to a second operation till it is in a thoroughly quiet state after the first is unjustifiable. With this reservation I would name one month after the first operation as the most suitable time for the second; earlier than this is unsafe, as the section may yield, or other complications may ensue. To wait longer is unnecessary in most cases, and possibly inexpedient as rendering the membrane harder to tear.

(5) *Exudation-products blocking the pupil.* These are the results of post-operative iritis, and vary greatly in density. Dissection is unsuitable for such cases, as the opening formed quickly closes again. I have tried Knapp's knife-needle for this form of secondary membrane, and found it most unsatisfactory, but must admit that the instrument supplied me was not of the best make. Iridotomy performed with Brudenel-Carter's intraocular scissors gives excellent results, with the proviso, however, that iritis always menaces the prognosis of a case.

Mr. Walter Jessop has recently shown me the ground-down cataract-knives he uses for dissection of such membranes. They are well worthy of a trial.

(6) *Dragging of the iris up into the section* has been sometimes included under secondary cataract. This condition is due either to severe iridocyclitis following operation or to a free escape of vitreous during the operation. In either case the prognosis is most unsatisfactory, and iridotomy is the best method of interference at our disposal. It is well not to expect too much from it.

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THE

Journal of Tropical Medicine

SEPTEMBER 15, 1902.

THE OLD AND NEW ARMY MEDICAL SCHOOLS.

THE transference of the Army Medical School from Netley to London marks a distinct epoch in the teaching of military medicine. Some condemn the step taken, and some enthusiastically approve of the decision to bring the young medical officers to the metropolis. The change is no doubt the outcome of recent events in South Africa ; it is really the result of the attempt made by ignorant persons to defame the medical officers of the Army engaged in the Boer war. If by their accusations and unjust criticisms, however, these hysterical persons have done good, the medical profession will be content to forgive them. The question is, what good has been done by pandering to the clamours of these would-be benefactors? We are not prepared to shut our ears to the arguments advanced by Sir Joseph Fayrer in his speech at the last prize distribution

at Netley. Sir Joseph pleaded the cause of the retention of Netley as the Military Medical School in terms which are difficult to refute, and many men agree with him.

It is not the first time the Netley school has been on the verge of being closed ; once, if not twice, it was only by the direct intervention of Queen Victoria that the Government laid aside their intention of iconoclastic dealings with Netley ; but at that time no substitute for the School was contemplated. The short-sighted Government of the time thought to save money by closing the School and stopping the special training of Army medical officers entirely. The Army medical officers of to-day have to thank Sir Joseph Fayrer for such a national calamity being averted ; for the cessation of special training for the Army medical officers is not one whit less absurd than it would be to turn lads accustomed to fire toy cannons at school to handle big guns without being trained. The institution fostered so carefully by Queen Victoria is, however, closed, and we must be prepared to help forward in every way possible the school under its new auspices.

The chief argument advanced for the new school is that it brings the cadets of the Army Medical Service in touch with the medical schools of the metropolis, and therefore in line with modern teaching. The young men, however, who join the Army Medical Service have only just left these schools ; they are acquainted with the most modern surgical and medical experiences of our universities, schools and hospitals ; they have but a few months previously served as either surgical dressers, clinical clerks, house surgeons or house physicians. It is not, therefore, a matter of urgency that they be in " touch " with the schools where they have studied and which they have just left. What they want is instruction in military medicine and surgery, military hygiene, hospital administration, and ambulance work. These are the subjects that specially distinguish the military officer from the civilian practitioner, and it is a question where these subjects can be best acquired. To our way of thinking, the magnificent clinical opportunities at Netley Hospital, the military regime there in vogue, and the

opportunity of acquiring a knowledge of hospital administration could be better acquired at Netley Hospital than in any other institution in Great Britain. Could Netley Hospital be brought to London with the School then there would be no need for discussion of the subject; for at Netley the medical officers have opportunities of becoming acquainted with tropical disease to an extent impossible in London.

However, the transference to London is an accomplished fact, and it behoves the civil medical profession to welcome their *confreres*. We are of opinion that the discipline followed at the Medical Staff College will have a beneficial influence in the metropolitan schools; and we would urge upon the civil schools the advantage of properly conducted residential quarters for their students. A model will be afforded by the Army Medical Staff College, and we have no doubt the authorities of the metropolitan medical schools will be willing and anxious to learn the methods of dealing with young medical men engaged in studying their profession. The Army Medical School, being in London, will also bring amongst us the able and distinguished men who are engaged as professors and instructors in the School. Their presence in London will strengthen the importance of our scientific meetings, and they will bring a wealth of experience to bear on many subjects which belong especially to their province. Their presence in London will also serve to keep the military and civil branches of our profession in more intimate touch, and will help to lessen the gulf which up to the present time has rather tended to widen than to close. We think, therefore, that whatever our opinions may be as to the expediency of moving the School, that professional, and thereby national, good will ensue; and we beg to welcome our military brethren to the metropolis, where we are sure the benefits to both the civil and military branches of medicine will be mutual.

The British Guiana Medical Annual for 1902.

ANKYLOSTOMIASIS: THE CATARRHAL CONDITION OF THE INTESTINAL TRACT.

By C. P. KENNARD, M.D.Edin., M.R.C.S.Eng.

In a paper in *B. G. M. A.* for 1899, I expressed the opinion that the great trouble in ankylostomiasis was the catarrhal condition and wasting of the intestinal tract and that the resulting indigestion was the chief cause of the anæmia in the chronic cases. I record this case as illustrating that; and it is also of interest as from the general appearance it has been diagnosed recently as chronic malarial cachexia, although it is very similar to the general run of cases so called, yet the malarial parasite has not been found in the blood during the year I had her under my observation, and the fever, when present, did not show a malarial periodicity.

Dulari, female, aged 30, E. I., ex *Lena*, 1900. October 5th, 1900, on arrival on the estate she was apparently well nourished, looking rather older than stated, nothing special about organs, stool contained ankylostome ova.

Between this and May she was in two or three times for slight ailments and was treated for the ankylostomes, being given calomel, grs. v., followed by ol. ricini and the day after three-hourly doses of thymol, grs. xxx., and ol. ricini afterwards if required.

May 7, 1901. — Admitted to hospital complaining of fever. Does not look strong, is rather anæmic. Spleen apparently not enlarged. Nothing special about her; for the next eleven days she had a daily irregular temperature varying between 102° and 99°, reaching normal on four days for a short interval; the skin was moist, there was no free perspiration; the blood examined on four separate occasions, no malarial parasite or pigment found, but some little red pigment, leucocytes about normal, in a very few unaltered R.B.C. were moving very small clear bodies, rod or pear shape, these bodies move *en masse* and do not show any protrusion of parts as the malarial parasite, they do not contain pigment; there were some dyspeptic symptoms, tendency to looseness of the bowels and pain in the stomach, but otherwise there was nothing definite about her.

May 20. — Says she is now all right, is rather anæmic. Spleen is enlarged, posteriorly just below the edges of the ribs. Discharged.

May 25. — Returns to hospital saying she has been getting fever since she has been out; is anæmic, looks worn, nothing definite about her; temperature 101°, dropping to normal the same day and rising to 99° the next.

Blood shows some increase of leucocytes; no malarial parasite or pigment. Given calomel and thymol.

May 29. — Looks better; tongue not so pale. Discharged.

June 3. — Returns, complaining of feeling weak and some pain in stomach; is anæmic.

June 4. — Stool examined, no ova found.

June 11. — An A. D. ovum found in stool; given thymol treatment.

June 17.—Tongue less pale; some abdominal pain.

June 21.—Is not improving; anæmic; complains of swinging in the head; spleen can now be felt nearly a handbreadth below ribs forward; is rather soft; no tenderness; complains of pain there sometimes; is not getting distinct fever but has frequent rises of temperature to 100°; no purging; has frequent pains in the stomach and umbilical region; abdominal walls lax and emaciated, on deep pressure, irregular lumps are felt and there is some tenderness; has lost flesh.

Blood shows no malarial parasite or pigment. Some clear small moving bodies in some R.B.C.'s; leucocytes about normal, but some increase of lymphocytes. A few days after, the temperature for seven days ran higher than usual, going up to 102° twice and keeping above normal; this, however, did not seem to affect her.

July 8.—She has improved and is looking better; not so anæmic. Discharged for light work and rations.

July 17.—Returns looking ill; very pale; wasted and anæmic looking; some slight puffiness round eyes and feet; tongue very pale; spleen now reaches below the umbilicus; some general tenderness of abdomen slightly over the spleen; abdominal walls slack; skin thin and dry; urine no albumen; says she has constant fever.

Blood examined, stain shows no malarial parasite or malarial pigment; R.B.C.'s vary in size; leucocytes about normal; no relative increase of lymphocytes or eosinophiles.

Stool rather offensive; containing a good deal of bile; no ova. The temperature varied daily for the next five days, running up usually in the evening to 103° or 100° and remaining about normal, it then became less, but occasionally ran from 100° to 101° for the next three weeks.

July 24.—Looking brighter; does not now complain of abdominal pain, but some difficulty in passing wind.

August 24.—Going on much as usual, gets a good deal of dyspepsia; frequently purging at night; some tenderness over abdomen generally; spleen enlarged and easily moved; abdomen slack; is emaciated; skin dry, thin and sallow; can find nothing special about organs. Blood very watery, no malarial parasite or malarial pigment; some variation in size of R.B.C.; leucocytes appear diminished.

Stool examined on the 22nd, to-day and on 30th, and no A.D. ova found, a good deal of undigested food, and to-day's show some small active amœba.

October 21.—Saw this woman to-day, she is still very thin but less anæmic; tongue not so pale, clean and rather raw looking, has some pain in the bowels and some tenderness on pressure; has diarrhœa occasionally; spleen much enlarged, not tender; nothing special about organs; no œdema of feet; she looks a little better than she did on August 30th.

January, 1902.—I understand this woman keeps much the same and nearly died a few days ago from an attack of diarrhœa.

Review.

THE AMERICAN ILLUSTRATED MEDICAL DICTIONARY. A New and Complete Dictionary of the Terms used in Medicine, Surgery, Dentistry, Pharmacy, Chemistry, and the kindred branches, with their Pronunciation, Derivation, and Definition, including much Collateral Information of an Encyclopædic Character. By W. A. Newman Dorland, A.M., M.D., Assistant Obstetrician to the University of Pennsylvania Hospital, Editor of the "American Pocket Medical Dictionary," Fellow of American Academy of Medicine. Together with new and elaborate Tables of Arteries, Muscles, Nerves, Veins, &c.; of Bacilli, Bacteria, Diplococci, Micrococci, Streptococci, Ptomaines and Leukomaines, Weights and Measures; Eponymic Tables of Diseases, Operations, Signs and Symptoms, Stains, Tests, Methods of Treatment, &c. With numerous illustrations and 24 coloured plates. London and Philadelphia: W. B. Saunders and Co., 1900.

This Dictionary was sent us for review. Perhaps the most convincing testimony to its value will be gathered when we state that the volume has never been allowed to be taken from our desk and that we find it of the greatest value, having occasion constantly to refer to it. Tropical ailments are so new an introduction to modern systematic medical literature, that omissions and errors might have been expected, but although we have made dozens of references to the subject of tropical diseases in the dictionary, we have on no occasion been disappointed. The number of technical names now in use are so manifold, especially in physiological-chemistry, in bacteriology, and even in ordinary medical, surgical, and gynecological work, that a dictionary of terms, &c., is an absolute necessity for medical men.

Correspondence.

ON THE LEUCOCYTES OF NORMAL BLOOD IN HOT CLIMATES.

To the Editor of the JOURNAL OF TROPICAL MEDICINE.

IT was pointed out in this Journal, September 2nd, 1901, by Mr. Horder, that in several European handbooks on pathology of blood, one met with average figures indicating the relative number of the different kinds of leucocytes in normal human blood which do not hold good in hot climates. As a result of his investigations he gives a proportion of about 5 per cent. of eosinophile leucocytes in Europeans and 15 per cent. in Chinese. When I observed this publication I happened to be engaged at Batavia (Java) in counting the relative numbers of the leucocytes of blood in a series of natives and Europeans. I can fully endorse Mr. Horder's conclusions. I found an average of 4.9 per cent. eosinophiles in Europeans, and 18.6 per cent. in natives. The number of lymphocytes is, compared to those of European blood, increased in both of them. The total number of leucocytes per m. M.³ here does not appear to differ widely from the average in Europe, 8,000—9,000. My results are published *in extenso* in *Geneeskundig tijdschrift voor Nederlandsch-Indië*, deel 42, afl. 3, blz. 211. Batavia, June, 1902.

Dr. C. D. OUMERANN.

News and Notes.

THE LONDON SCHOOL OF TROPICAL MEDICINE.

MR. BOMANJI DINSHAW PETIT has contributed a lakh (100,000) of Rupees to the funds of the London School of Tropical Medicine. The gift was made in response to the mission and influence of Sir Francis Lovell, during his journey to the Tropics on behalf of the School. At first the donation was anonymous, but Mr. Bomanji Petit has, by request, allowed his name to be published. In a letter to Sir Francis Lovell, dated August 14th, Mr. Bomanji Petit writes: "As you want my permission to declare my donation of Rupees, one lakh, to the London School of Tropical Medicine, to the public, and also to allowing my name being associated with the new building, I give my consent to do so." Mr. Bomanji Dinshaw Petit is the surviving son of the late Sir Dinshaw Maneckji Petit, Bart. The distinguished Parsee family of Petit has long been well known for its public-spirited generosity, and the present gift bears testimony that that valuable characteristic is still being maintained as a family tradition. It is to be hoped that His Majesty, on the occasion of the approaching Delhi Durbar, will signify the nation's appreciation of Mr. Bomanji Petit's many acts of philanthropy in an official manner; and were the distinction conferred upon his father bestowed upon his illustrious son, the many peoples and races interested in combating disease in the British Possessions would rejoice to know that one who has done so much for the alleviation of suffering was held in esteem by his Sovereign.

We have received the subjoined circular:—

PROPOSED JOURNAL FOR THE ARMY MEDICAL SERVICES.

It is believed that the establishment of a journal devoted to matters of professional and scientific interest would be generally welcomed by officers of the Army Medical Services, as affording to them advantages similar to those already enjoyed by other branches of the British Army, and by the medical services of Continental Powers.

The proposed journal would to a great extent take the place of the present appendices of the Army Medical Department Report, and would embrace the following items:—

- (1) Original articles written by officers belonging to the Army Medical Services, and others.
- (2) Bibliographical notes on articles of importance and interest to the military services.
- (3) Reprints and translations from military, medical, and other journals.
- (4) Official gazettes, and official information generally, bearing upon the Army Medical Services.

It is hoped that a journal conducted upon these lines will enable Medical Officers to keep in touch not only with what is going on in the British Service, but with the advances and changes that are being made in other armies.

The journal will be conducted and edited under the supervision of a committee representative of the Headquarters Staff, the Medical Staff College, and the

Advisory Board of Army Medical Services, and to this committee Officers who have made special studies of any subject are requested to give their names as referees on that particular subject. It is also hoped that those who have a knowledge of foreign languages, and are capable of undertaking the work of reviewing and extracting information from foreign publications, will send in their names for work of that description in connection with the journal.

The Director-General would be glad if Officers would let him know their views as to the general management and contents of the proposed journal. It should, however, be stated that its pages will not be open to controversial correspondence, or to items of social or personal interest, other than what is official.

In conclusion, the Director-General ventures to hope that there will be no hesitation in supporting this effort to maintain a high standard of professional and scientific attainment in the Army Medical Services, and he would be obliged if Officers will at their earliest convenience state whether they are willing to support the journal, it being understood that the annual subscription will not in any case exceed £1.

18, VICTORIA STREET, S.W.

July, 1902.

[We welcome the proposed addition to our literature, and we congratulate the public-spirited action of the Army Medical Staff.—ED. J. T. M.]

Current Literature.

THE DIAGNOSIS OF MALARIA.—In the *Medical Record* for April 5th, 1902, Dr. Ford emphasises the necessity for examinations of the blood in making a diagnosis of malaria. In one series of 7,000 cases suffering from various ailments, malarial fever was stated to exist as the cause of admission, or as a complication thereof, in from 54 to 55 per cent. of all cases, the maximum being in January and February of each year, the minimum in July and August. Upon the arrival of a competent pathologist the average fell to about 10 per cent., and remained in that vicinity until the pathologist departed, when it immediately rose again. The diagnosis reached during the time of the pathologist's stay had been by means of the microscope only, due allowance being made for the recent use of antiperiodics, spontaneous convalescence, &c. The diagnosis reached at other times was clinical.

BROOKE'S PASTE IN SKIN DISEASES (Dreyer, *Dermat. Zeit.*, 1902, p. 19).—This paste has the following composition:—

| | |
|---------------------------------|-----------------|
| Oleate of Mercury (5 per cent.) | 28 parts. |
| Oxide of zinc and starch | ... aa 7 " |
| White vaseline... | ... 14 " |
| Ichthyol | ... 1-2 " |
| Salicylic acid | ... 1-1/2 part. |
| Oil of lavender | ... q.s. |

The author has used it with success in sycosis furunculosis. A case of gangrene of the penis after cocaine injection was cured in three weeks. Syphilitic

ulcers of the vagina were cured in eight days. Good results were obtained in inflamed hæmorrhoids, and eczema, herpes progenitalis, scrofuloderma, hard chancre, syphilitic ulcers (except malignant). The author attributes the success to the combined effect of the mercury, salicylic acid, and ichthyol.

PROTEOSOMA MALARIA IN SPARROWS.—Dr. W. N. Berkeley submitted specimens and described the results of some work he had done in September, 1901, on the subject of malaria in birds. It was easy in the Bronx Borough to find English sparrows (*Passer domesticus*) infected with malarial parasites corresponding in all morphological characters with the proteosoma of Labbé.

Dr. Berkeley had examined many hundred birds, of which 75 per cent. were infected slightly and 15 per cent. severely. The latter, though apparently well when in the open, sickened rapidly in confinement, refusing to eat, and sitting with ruffled feathers in a corner of the cage.

The temperature of one bird reached 108.8° F., though the speaker would not venture to say that this figure represented an unusually high fever in birds. The heart-blood of one bird, examined *post mortem*, showed many red cells invaded by five to seven sporocytes, apparently a multiple infection. He had not found similar observations recorded in the literature, and was not able to claim that this form of infection was of proteosomal nature.—*New York Pathological Society. Stated Meeting, held April 9th, 1902.*

MALARIA IN TURKESTAN.—In the *Roussky Vrach*, June 15th, Dr. S. A. Mark states that two groups of malarial plasmodia exist in Turkestan, viz., the parasites of tropical malaria and the parasites of more temperate climates—quartan and tertian. Of 161 cases observed, the tropical parasites were eight times more prevalent than the temperate varieties in low-lying places with high temperature. In high ground, however, the temperate parasites prevailed. When typhoid fever develops in malarial patients, the growth of the plasmodium seems to be inhibited for the time; when the attack is over, however, the malarial poisoning gains the upper hand. The same is true in the case of most diseases of a nature intercurrent in malarial patients. Tuberculosis assumes a very rapid course in persons suffering from malarial disease.

THE TREATMENT OF YELLOW FEVER.—After an historical review, James Carroll considers this question in the light of our present knowledge. He believes that in previously healthy subjects, free from organic lesions, the mortality can be practically reduced to zero. The primary indication is to remove the toxin. This is best done through the kidneys, aided by a moderately free action of the skin and moderate depletion by saline cathartics. Hot mustard foot-baths, sinapisms to the stomach, cold cloths to the head, saline enemas with sodium sulphate solution, are all indicated. For the first three days only milk with vichy or lime water should be given. Iced champagne is permissible. Other treatment follows along symptomatic lines. The author suggests that urea taken by the mouth or injected under the skin may serve as a

valuable diuretic.—*Journal of the American Medical Association, July 19th, 1902.*

THE SO-CALLED "SPOTTED FEVER" OF THE ROCKY MOUNTAINS.—In a preliminary report to the Montana State Board of Health, L. B. Wilson and W. M. Chowning state that they could find in this disease no bacterium standing in a positive etiological relation. Staphylococci of different varieties were found in the skin, and *B. coli communis* in cultures from the spleen. The latter organ showed in one case a special anaerobic putrefactive bacillus. So also several protozoa were present, some of which apparently found their habitat in the red blood-cells. The prevailing protozoon resembled the pyrosoma bigeminum of Texas fever. Apparently, spotted fever belongs rather to the latter type of disease than to the malaria group. The most probable theory as to the mode of infection ascribes the latter to tick-bites. The extreme isolation of cases of spotted fever, their occasional development in localities removed many miles from the site of any previous case, and the long period existing between the death or convalescence of the last case of any one year before the development of the first case in the following year, would point to the possibility of the red blood-cells of some one of the lower, warm-blooded animals being the normal habitat of the parasitic protozoon in that stage of its life cycle not passed within the body of some arachnid. Of the animals within the infected region, the common gray gopher would probably best fulfil the conditions of such a parasitism.—*Journal of the American Medical Association, July 19th, 1902.*

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- 2.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.
- 3.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.
- 4.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.
- 5.—Correspondents should look for replies under the heading "Answers to Correspondents."

The Journal of Tropical Medicine.

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Original Communications.

SLEEPING-SICKNESS AND FILARIA PERSTANS IN BUSOGA AND ITS NEIGHBOURHOOD, UGANDA PROTECTORATE.

With Map and Diagrams—see plate.

By AUBREY HODGES, M.D.Lond

Medical Officer, Uganda Protectorate.

THE present paper is founded (1) on notes and observations made on an expedition of about a month's duration, undertaken in February and March, 1902, which included the northern shores of Lake Victoria Nyanja and the adjacent islands, and during which 375 case of sleeping-sickness were examined; (2) on notes of 77 cases taken at Jinja, the Government station in Busoga, in which province lies the centre of the present epidemic; (3) on examination of the apparently sound wherever possible; and (4) on first-hand information from chiefs and others of various localities.

The first aim of the expedition was to reach the extreme outskirts of the epidemic area, where it appeared to the writer that the conditions, especially with regard to filaria perstans, could most profitably be studied. This aim, however, was not to be attained, as the epidemic had already spread beyond the legitimate scope of the expedition; nor was it possible, as had been hoped, to hear of a single island as yet unattacked by the disease. But it will be seen from Table II. (p. 297), and the accompanying map, that a line or area of lesser intensity was reached in Kavirondo, the facts gathered in which, if borne out by further inquiry, may prove to be of some importance. These facts relate to the presence in the blood of both sick and sound of filaria perstans and, taken with other facts noted during the expedition and afterwards, would seem to emphasise the importance of early investigation over

a very large area, which grows larger day by day as the epidemic spreads, in order that the conditions obtaining at and beyond its borders may be ascertained.

Sleeping-sickness was found to be most prevalent in the central and south-central portions of Busoga and on the neighbouring islands. In some places many hundreds had died and thousands were found to be suffering from all stages of the disease.

History of the epidemic (Native report).—The general opinion among the Busoga and neighbouring natives is that the disease came from Wakolis (a central district in Busoga). The story in Wakolis itself is that a long time ago (1892) a small number of Masai (Wakwafi from Njemps) came and settled at Wakolis; these people had many sick and at last determined, on that account, to return to their own country. As a matter of fact, they returned in batches, the last some time in 1896. About the time they were leaving, the Busoga began to be attacked by a disease which they at first thought was plague (Kaumpuli), but when they did not soon die, and they saw them nodding, they said, "This is the disease the Masai had," and they suggested that it originated with these Masai or Wakwafi. It is strange, if this were so, that nothing has been heard of sleeping-sickness in the district of Baringo (Njemps) or in Masailand, but it would seem not improbable that the Busoga first noticed the disease in their country about that time, though there is doubt as to whether the disease from which the Masai suffered was of this nature. The Busoga are of a rather low order of intelligence and have little faculty of memory, but there is general agreement that cases of sleeping-sickness began to get numerous in Wakolis country after the Sudanese mutineers passed through in 1897, and also about the time of the passage of the big caravan of the 27th Bombay Light Infantry, early in 1898.

Another story, not met with by the writer, and

denied to him by several of the older chiefs, including a son of the Wakoli of that time whose land adjoined Wakolis, is that the disease came from Buda (south of Buganda proper and adjoining German East Africa) and spread along the west shore of Lake Victoria to Busoga. In that case sleeping-sickness would almost certainly have been noticed in Buda and among the more intelligent Buganda, who have been since 1890 in contact with Europeans, and it is possible that this account may refer to plague (Kaumpuli) which is endemic in Buda and the neighbouring German territory, and with which sleeping-sickness was at first, and is sometimes still, confused.

There is also a third story, an important one if true, which tells of an epidemic about 1887 in Bunyoro and Toro (west of Buganda and adjoining the Congo Free State), but this at present rests on the testimony of one native only, and has as yet received no confirmation.

The following is the *duration of the epidemic in various districts according to local report* (March, 1902):—

Wakolis, five and a half to six years; Buvuma Island, two years and four months at least; Jinja, Lubas and Naniumbas, one year; Nyala, nine months; Sigula Island, six months (*vide* Map).

In considering the length of the above periods it should be remembered that the natives, attempting a primeval form of isolation, and until the numbers become too great, are accustomed simply to drive away into the bush such persons as are suspected of having a communicable and fatal disease, such as small-pox, plague or sleeping-sickness, and that they have hitherto hidden their sick from the sight of Europeans, probably from the unfounded fear that they themselves may be likewise driven away or disturbed on account of sickness among their people. Even now (at the time of the expedition), in spite of its prevalence, all signs of sleeping-sickness might easily escape a casual observer passing through the country. It is only when the poor creatures are stimulated by the hope of medicine or cure, that the sad crowds emerge from their huts and enclosures, or from their improvised shelters in the jungle.

Enumeration of symptoms (from 452 cases).—The *enlargement of superficial glands* appears to be certainly general, practically constant, and probably very early in manifestation. Any one group of glands may be more or less affected than the others, but those in the neck are the most often swollen, though the preponderance is not very marked. The enlargement is usually pronounced in children, may sometimes be very slight, especially in old people, but is seldom altogether absent. Also in the few *post mortems* made, the mediastinal, mesenteric and other lymph glands have been found enlarged.

No specific rash was detected, and the vast majority of cases were without rash of any kind, but *harsh and dry or scaly skin* is common, especially in advanced cases.

Itching and scratch-marks are very often met with, but are common in healthy natives, and

scratches or scales may be masked or obliterated by inunction of the skin. The *pruritus* of sleeping-sickness is, nevertheless, a very constant symptom; it appears to be general and is very persistent and troublesome, being to some extent comparable clinically with the itching of jaundice.

Tongue tremor is very constant and early, and general tremor is frequently met with in well-advanced cases. Tremor of the tongue is, not very uncommonly in young people marked or replaced by jerky *choreiform twitches*, which sometimes extend to the mouth, face and hands, and occasionally to the whole body. These phenomena seem generally to occur in the earlier stages, and the ages most affected to coincide with those most affected by true chorea. No heart mischief has been detected.

A peculiar *thickness of speech or mumbling* has been noted in many cases, and in a few stammering. This last, when it is present, seems to occur very early, and might therefore occasionally be an aid in diagnosis.

Cerebral excitement or mania has been met with or reported in comparatively few cases, but there is reason to believe that in cases watched throughout their course it would be found to be less uncommon.

Patients sometimes complain of *giddiness and falling down* in the early stages, and *convulsions* of an epileptiform nature have been seen at a late stage in one case.

Left-sided *ptosis*, very marked, but clearing up entirely in about a fortnight, was seen in one case, and muttering delirium towards the last was noted in two cases.

Most patients complain of *pain* about the body and limbs, but the most constant pain is *headache*, which is often the most distressing symptom to the patient, occurs very early, and is probably, as a rule, persistent throughout the disease. It not infrequently extends to the occiput and back of the neck. Pains in the chest and limbs are often complained of, but muscular tenderness has not been found, and although the muscles become extremely flabby and weak, nothing like paralysis has been detected.

Muscular weakness is invariable and progressive, but is often not complained of in the later stages. The *staggering gait* which it causes sooner or later has sometimes seemed to resemble alcoholic inco-ordination, sometimes pseudo-hypertrophic paralysis and sometimes locomotor ataxy, but as a general rule it is atypical.

Dulness, lethargy or somnolence is sooner or later present, as described in text-books, but may be preceded by a period of headache, with tongue tremor, glandular enlargement and perhaps pruritus, and sometimes cerebral excitement or mania. During cerebral excitement lethargy is, of course, absent, and in the early stages, during conversation, attention to things of interest, or physical exertion, the patient's appearance may be quite normal for considerable periods.

It is uncommon to find *pyrexia* at a single examination, the temperature being usually subnormal, but all cases watched for any length of time have

shown some febrile disturbance, and this has occurred in one or two cases apparently at the very beginning of the disease.

Emaciation is rare so long as food is obtainable, but is sometimes very extreme towards the last.

Analysis of symptoms.—The statement made above, that the enlargement of the superficial glands is general, would seem to be justified by the following figures.

TABLE I.

ENLARGEMENT OF SUPERFICIAL GLANDS.

| GENERAL | NECK ONLY | AXILLÆ ONLY | GROINS ONLY | NONE IN NECK | NONE PERCEPTIBLE |
|-----------|-----------|-------------|-------------|--------------|------------------|
| No. | No. | No. | No. | No. | No. |
| 366 | 15 | 3 | 1 | 18 | 7 |
| per cent. | per cent. | per cent. | per cent. | per cent. | per cent. |
| 80.9 | 3.3 | 0.6 | 0.2 | 3.9 | 1.5 |

The cases classed as general are those in which the chief groups, cervical, axillary and femoro-inguinal, were all involved, with or without the submental, post-aural, sub-occipital and supra-condylar groups. It will be apparent that the glands of the axillæ and groins are very commonly affected, and it may be added that in nearly the same degree of frequency enlarged glands may be found in all the groups. The cervical, however, are the most constantly swollen. Often one side of the neck is more affected than the other, and it is curious that out of 86 cases in which the difference was noted, the left side was more affected in 61 and the right only in 25. The femoral glands are often more palpably enlarged than the inguinal.

The swollen glands are not visibly enlarged and are rarely tender, at least when the disease is fairly established, though it is possible that a subacute adenitis may sometimes be an initial or prodromal symptom. For the rest, they are of a moderate hardness, discrete and freely movable under the skin, and there seems to be no tendency to suppuration.

From the small number of cases, only $1\frac{1}{2}$ per cent., in which there was no perceptible glandular swelling, it may be taken that this symptom is practically constant; but it must not be forgotten that glandular enlargement without visible cause is much more common among the natives here than in Europe, for instance; and that vermin in clothes or hair, small sores or chiggers on hands and feet, syphilis, itch and other skin eruptions will account for a good many cases of superficial adenitis, which, however, will often be localised by the lesion to which it is due.

The above considerations also hold in the case of pruritus, which is an only slightly less prevalent symptom, having occurred in 414 cases, or 91.5 per cent.

Skin eruptions were noted in 38 cases, 8.4 per cent. Of these 20 were itch, or the common papulo-pustular dermatitis of the country, which is curable by

sulphur; 12 were described as finely papular, and were generally situated on the hands and arms, sometimes also on the chest, back and legs; one was described as flat, shiny papules on hands and arms; one as fine and flattish papules, inclined to be scaly, on chest, arms and hands, with eczema between the fingers, "resembling itch"; and four as "a few scattered papules" in various regions.

Choreiform movements or irregular twitchings were noted in 57 cases, or 12.6 per cent., 34 being males and 26 females. The preponderance of males is less than it appears, for the total numbers of males and females being 292 and 260, the respective percentages work out to 11.5 and nearly 9. As regards age,* 47, or 82.4 per cent., of the cases were under 15 years, and about three-fourths of these, again, were under 10 years, while the total number of patients under 15 years being 144, the percentage of these symptoms among them (children) was as high as 32.6. It is possible, however, that in taking these cases a certain amount of unconscious selection was used, for, in taking perhaps 40 out of several hundreds, it is difficult to omit such as are so visibly striking and interesting as these often are. The oldest female affected was 45 and the oldest male 20.

This involuntary jerky twitching is sometimes very slight, and may be confined to the tongue, while its existence in a given case might often be a matter of opinion. Only such cases as seemed well marked were noted, and in many instances it is sufficiently characteristic. It is most often to be seen in the tongue and lips, and seems to extend from there in varying degrees. It is apparently distinct from the tremor which is in greater or less degree so universal and which becomes more marked as the disease progresses, whereas choreiform movements, so far as the writer's observation goes, tend to disappear. There is want of muscular control, as sometimes shown in inability to put out the tongue at will; then, perhaps, it is protruded with a sudden jerk, when it is either immediately withdrawn in the same manner, or is clasped tightly by the oral muscles. The movements are increased or brought into notice by effort, they seem seldom to become general, and no case was seen in which they were in any sense violent. The muscles of the upper part of the face seem less frequently or less prominently involved than in true chorea, and consequently the grimacing so characteristic in this disease is much less marked. The affection may apparently be, so far as it goes, unilateral.

Choreiform movements were noted as follows:—

Tongue only, four cases; tongue and face, 26; tongue, face and hands, 12; tongue, face, hands and arms, 11; general, 4.

One cannot help recalling, in connection with this interesting symptom, the old theory of "clouds of minute emboli," suggested, I believe, by Dr. Hughlings Jackson, to explain true chorea, and wondering whether perstans embryos could act as minute emboli or inflammatory centres, though the phenomena

* Apparent ages only are given, as no native has any idea of his age.

are no doubt due to the brain changes described by Dr. Mott, however these may be caused.

The thickness of utterance or mumbling speech mentioned above is probably due to the tongue affection. A patient in the early stages with this affection of speech, or stammering, with staggering gait, tremor and perhaps cerebral excitement, might easily be supposed to be under the influence of alcohol. Indeed, one of the earlier cases seen gave at first that impression.

Cerebral disturbance, as distinguished from the stupor of later stages, was noted in 14 cases, 12 males and two females. In 11 it was described as mania and in three as cerebral excitement. It sometimes takes the form of exhilaration. The symptom appears to be usually a comparatively early one, and may possibly sometimes be initial, at least so far as the graver phenomena are concerned, but it may occur when the disease is well-developed, and may be present up to the last. There is naturally a difficulty in obtaining information from these cases, and in three the length of illness was not ascertained. In two it was given as seven and ten days, and the rest were stated to have been ill from one to three months. Of these one was said to have been mad for two months, *i.e.*, from the beginning; in one mania supervened after he had been for nearly a month under observation, and had been ill for, probably, at least three months; and in one who was seen in a late stage, it was said to have begun very early and was afterwards reported to have persisted till the end.

With regard to the typical aspect of the patient with sleeping-sickness, there is probably nothing to be added to the graphic description given in Manson's "Tropical Diseases." But, as has been already pointed out, in the early stage the patient's appearance may be perfectly normal during what may be termed his "lucid intervals," when he may be seen talking, laughing or working like other people. These intervals may be of considerable duration at first, and there is reason to believe that it will be found that there is frequently a more or less prolonged period of malaise before any sign of lethargy becomes apparent. One must not, therefore, expect every case of sleeping-sickness to present a definite aspect, or early cases may be overlooked. When the patient is perfectly quiescent, however, even if there is no other visible sign, there is often a peculiarly sad look of aloofness and distress, such as may be seen in a stricken animal.

Most probably this facial expression is due to headache, which, as has been said above, is one of the earliest, most constant, and most persistent symptoms.

Duration of the illness.—It is difficult to arrive at a just conclusion without watching many cases throughout their course, and it is very difficult, in the present state of our knowledge, to fix, in a given case, with any preciseness, the date of commencement of the disease. But it is probable that the average duration of life among natives left to themselves, from the time the symptoms are first recognised by them, is from three to four months. The longest

period of illness given by patients at the time of examination was nine months, and a good many stated the length of their illness to be six months, but their ideas of time are very crude. A chief's wife, in whose case the time given can be taken as approximately correct, gave the length of her illness as nine months, and her death was reported about a month afterwards. It is certain that patients may seem to be scarcely ill, and die in a month's time.

The natives are generally agreed that the disease is invariable fatal.

The infection and spread of the disease.—From general observation of the epidemic and its extreme prevalence near the shores of the lake and on the islands, it would appear probable that the infection, whether it be *F. perstans* or no, may be derived either from the water or something connected therewith; either directly or through some mosquito or blood-sucking insect.

(1) *Water.*—It is difficult to imagine infection pervading such a vast body of open water as Lake Victoria, though it might inhabit the swamps, pools and streams connected with the lake. But people who derive their water-supply from sources which would seem to be practically above suspicion, such as the rapidly running stream of the Victoria Nile, do not seem to be in any way immune. Also among a large number of people at Lubas, who got their water from a practically fine rock-spring, with no stagnant dipping-pool attached, which issues from somewhere near the middle of the side of a declivity of about 600 feet, *F. perstans* was almost invariably found and sleeping-sickness was extremely prevalent. It appears, therefore, most unlikely that the infection is contained in the water itself.

(2) *Fish.*—What seems at present at least possible is that the extensive traffic in fish, which is carried from the coast and islands over nearly the whole of Busoga and a great part of Kavirondo, may be in some way connected with the spread of the epidemic, more probably, perhaps, through the inter-communication involved than the fish carried. This traffic extends as far as the Mpologomo River, which is the northern boundary of Busoga, but not to the further side, where, according to unanimous reports of local chiefs, there is as yet no sleeping-sickness,* and where it seems fairly certain that at least it does not exist in epidemic form. From the river itself, however, a large quantity of fish is taken, of which several kinds are reported by natives to be identical with those carried from the lake. In north-west Busoga, where there is very little traffic with the lake, there are said to be very few cases of sleeping-sickness. It may be mentioned that the Busoga scarcely ever cook animal food thoroughly, while in times of scarcity they will eat any dead animal, or even insect, which they may pick up.

Taking native report as approximately correct, the extremely gradual spread of the epidemic in its first few years is worthy of note, and may be supposed to point to the necessity of a long period of develop-

* It seemed doubtful whether there might not be a few cases among the Wadema.

TABLE II.
SLEEPING-SICKNESS AND FILARIA PERSTANS.

| RESIDENCE | RACE | SLEEPING SICKNESS | F.P. | PER CENT. | SOUND CASES | F.P. | PER CENT. | | | | |
|-----------------|--------------------|-------------------|-----------------|-----------------|----------------|-----------------|-----------------|-----------------|----------------|------|------|
| Jinja | Basoga (residents) | { over 7 years | 16 | 15 | 93.7 | { over 7 years | 52 | 41 | 78.8 | | |
| | | { under 7 years | 4 | 2 | 50. | { under 7 years | 11 | 1 | 9. | | |
| | | { Total | 20 | 17 | 85. | { Total | 63 | 42 | 66.6 | | |
| | (prisoners) | { over 7 years | — | — | — | { over 7 years | 38 | 32 | 84.2 | | |
| | | Baganda (police) | { over 7 years | — | — | — | { over 7 years | 40 | 26 | 65. | |
| | | | { over 7 years | 3 | 3 | 100. | { over 7 years | — | — | — | |
| | (residents) | | { under 7 years | 1 | 1 | 100. | { under 7 years | 7 | 5 | 71.4 | |
| | | { Total | 4 | 4 | 100. | { Total | — | — | — | | |
| | | Sudanese | { over 7 years | 1 | 0 | 0. | { over 7 years | 6 | 3 | 50. | |
| | { under 7 years | | — | — | — | { under 7 years | 2 | 0 | 0. | | |
| | { Total | | 1 | 0 | 0. | { Total | 8 | 3 | 37.5 | | |
| | Iganga | Lendus | { over 7 years | — | — | — | { over 7 years | 2 | 2 | 100. | |
| { under 7 years | | | 1 | 1 | 100. | { under 7 years | — | — | — | | |
| { Total | | | 1 | 1 | 100. | { Total | — | — | — | | |
| Bakedi | | { over 7 years | 1 | 1 | 100. | { over 7 years | — | — | — | | |
| | | Basoga | { over 7 years | 1 | 1 | 100. | { over 7 years | 2 | 2 | 100. | |
| | | | { over 7 years | 1 | 1 | 100. | { over 7 years | — | — | — | |
| Kayangas | | | { over 7 years | 1 | 1 | 100. | { over 7 years | 2 | 2 | 100. | |
| | | { over 7 years | 8 | 8 | 100. | { over 7 years | — | — | — | | |
| | | { under 7 years | 1 | 1 | 100. | { under 7 years | 1 | 1 | 100. | | |
| Wakolis | | { Total | 9 | 9 | 100. | { Total | — | — | — | | |
| | | Banyoro | { over 7 years | — | — | — | { over 7 years | 1 | 1 | 100. | |
| | | | Basogo | { over 7 years | 4 | 4 | 100. | { over 7 years | — | — | — |
| Kairanyas | { over 7 years | | | 4 | 4 | 100. | { over 7 years | — | — | — | |
| | Nambogwe | { over 7 years | | 3 | 3 | 100. | { over 7 years | 8 | 5 | 62.5 | |
| | | Gabras | { over 7 years | 1 | 1 | 100. | { over 7 years | — | — | — | |
| | | | Tabingwas | { over 7 years | 4 | 4 | 100. | { over 7 years | — | — | — |
| | Kisikis | | | { over 7 years | 5 | 2 | 40. | { over 7 years | — | — | — |
| | | Banda | | { under 7 years | 3 | 0 | 0. | { under 7 years | 18 | 10 | 76.9 |
| | | | { Total | 8 | 2 | 25. | { Total | — | — | — | |
| | Lubas | | { over 7 years | 181 | 172 | 95. | { over 7 years | 34 | 31 | 91.1 | |
| | | { under 7 years | 25 | 20 | 80. | { under 7 years | 11 | 7 | 63.6 | | |
| | | { Total | 206 | 192 | 93.2 | { Total | 45 | 38 | 84.4 | | |
| | Nanumbas | Bavuma | { over 7 years | 30 | 29 | 96.6 | { over 7 years | — | — | — | |
| | | | { under 7 years | 12 | 7 | 58.3 | { under 7 years | 12 | 10 | 83.4 | |
| { Total | | | 42 | 36 | 85.7 | { Total | — | — | — | | |
| Iagusi I. | | { over 7 years | 48 | 42 | 87.5 | { over 7 years | 13 | 11 | 84.6 | | |
| | | Buvuma I. | { under 7 years | 3 | 2 | 66.6 | { under 7 years | — | — | — | |
| | | | { Total | 51 | 44 | 86.2 | { Total | 58 | 44 | 75.8 | |
| Baganda* | | | { over 7 years | 0 | 0 | 0. | { over 7 years | 22 | 21 | 95.4 | |
| | | { under 7 years | 0 | 0 | 0. | { under 7 years | 6 | 1 | 16.6 | | |
| | | { Total | 0 | 0 | 0. | { Total | 28 | 22 | 78.5 | | |
| Bugaya I. | | Bavuma | { over 7 years | — | — | — | { over 7 years | 25 | 17 | 68. | |
| | | | Wima I. | { over 7 years | — | — | — | { over 7 years | 9 | 6 | 66.6 |
| | | | | Damba I. | { over 7 years | — | — | — | { over 7 years | 11 | 8 |
| | Mengo | { over 7 years | | | 3 | 3 | 100. | { over 7 years | — | — | — |
| | | Kyagwe | { over 7 years | | 5 | 4 | 80. | { over 7 years | — | — | — |
| | | | { under 7 years | 1 | 1 | 100. | { under 7 years | 21 | 17 | 80.9 | |
| | { Total | | 6 | 5 | 83.3 | { Total | — | — | — | | |
| | Njala | { over 7 years | 29 | 1 | 3.4 | { over 7 years | 46 | 1 | 2.1 | | |
| | | { under 7 years | 5 | 2 | 40. | { under 7 years | 4 | 0 | 0. | | |
| | | { Total | 34 | 3 | 8.8 | { Total | 50 | 1 | 2. | | |
| | Sigulu I. | Kavirondo | { over 7 years | 41 | 3 | 7.3 | { over 7 years | 42 | 1 | 2.3 | |
| | | | { under 7 years | 4 | 0 | 0. | { under 7 years | 10 | 1 | 10. | |
| { Total | | | 45 | 3 | 6.7 | { Total | 52 | 2 | 3.8 | | |
| Budi | | { over 7 years | 2 | 2 | 100. | { over 7 years | — | — | — | | |
| | | Igagas | { over 7 years | — | — | — | { over 7 years | 5 | 1 | 20. | |
| | | | Dungas | { over 7 years | — | — | — | { over 7 years | 3 | 0 | 0. |
| Bakedi | | | | { over 7 years | — | — | — | { over 7 years | 34 | 29 | 90.6 |
| | | Various | | { over 7 years | — | — | — | { over 7 years | 7 | 1 | 14.2 |
| | | | Swahili | { over 7 years | 1 | 1 | 100. | { over 7 years | — | — | — |
| Europeans | | | | { over 7 years | — | — | — | { over 7 years | 10 | 1 | 10. |
| | | Total | | { over 7 years | 393 | 305 | 77.6 | { over 7 years | 523 | 329 | 62.9 |
| | | | { under 7 years | 59 | 35 | 61. | { under 7 years | 44 | 10 | 22.7 | |
| { Grand Total | 452 | | 341 | 75.4 | { Grand Total | 567 | 339 | 59.8 | | | |

* Out of about 400 Baganda who had been resident in Buvuma 16 months at the date of examination there had been one case of Sleeping-Sickness which had already proved fatal.

ment either for the infection or the disease caused by it, and to the supervention of some special conditions, either internal or external, to account for its later rapid spread among the population. A severe famine in 1899-1890, and several periods of unusual scarcity since, together with a recent severe and wide-spread epidemic of small-pox, have probably acted as predisposing causes of the acceleration of its progress.

Supposing that *F. perstans* were shown to be the cause of sleeping-sickness, it is, of course, judging from analogy, more likely that the embryo finds its intermediate host directly in some blood-sucking insect, but, until its development has been demonstrated, the possibility cannot be excluded that certain of the fish themselves may carry it, and that Dr. Manson's original theory with regard to *F. nocturna* may prove to be correct in the case of *F. perstans*. Nor can the possibility of its being carried in this way be at present disregarded, whatever the infection may be.

Filaria perstans.—The general results of examination of the blood of sleeping-sickness patients and others for *F. perstans*, will be found in Table II., and the extent of distribution of *F. perstans* and of sleeping-sickness, so far as could be determined by observation and enquiry, is shown on the accompanying map.

The most striking fact shown by the table is the very low percentage of filariæ found both in sick and sound in Kavirondo, as compared with the very high percentage in other parts, even where sleeping-sickness is stated to be absent. Out of 81 Kavirondo sick, *F. perstans* was found in only eight, or 9·8 per cent., and out of 110 sound, it was found in only four, or 3·6 per cent.; whereas, taking all other cases except Europeans, *perstans* was found in 333 out of 371 sick, or 89·7 per cent., and in 337 out of 457 sound, or 73·8 per cent. An attempt is made to show on the map how the areas of sleeping-sickness and of *F. perstans* overlap one another, the former to the east (Kavirondo) and the latter to the north (Bukedi), where, as will be seen from the table, the percentage of filariæ was as high as 90·6, being 94·4 in the neighbourhood of Mount Elgon and 71·3 in the neighbourhood of Lake Kioga. At present it can only be said that the point calls for further investigation. It would be hasty to draw definite conclusions therefrom as to the causal association of *F. perstans* with sleeping-sickness, since the conditions which determine the presence of the embryo in the peripheral circulation are as yet entirely unknown, and, moreover, it will be noticed that in all cases, broadly speaking, the filaria was found in a rather higher percentage of the sick than of the sound.

So far as could be ascertained at the time it was visited (March, 1902), western Kavirondo, though by no means the limit, was somewhere near the eastern boundary of the epidemic area, and though the sick were numerous, there had not been so many deaths, and fewer were seen in late stages of the disease.

Table II. also appears to show that *F. perstans* is less common, or less numerous, in the peripheral circulation at any rate, in young children than in

older persons and adults, with or without sleeping-sickness. An arbitrary division for the sake of statistics has been made at the age of 7, but it is probable that the younger the child the less likely is the filaria to be found. The writer has not yet seen either sleeping-sickness or *F. perstans* in a child under 1 year old, though four cases of the former were seen at about 1½ years, in two of which the filaria was found. The earliest age at which *F. perstans* was found in a sound person was 2½ years.

A point that is worthy of further investigation is that glandular enlargement and pruritus, two symptoms almost universally present in sleeping-sickness, appear to be more common in persons harbouring *F. perstans* than in those who are free. The writer was first struck with the fact that this might be so at the examination of 38 prisoners on February 8th, last, when the filaria was found in 32, of whom no less than 19 showed general swelling, while six more had enlarged glands in various situations, making a total of 25; also 16, not including two who had skin eruptions, complained of itching of the skin. On the other hand, among the six who had no filariæ there was found neither itching nor glandular enlargement. Three of these filariated cases with itching and glandular enlargement subsequently developed definite signs of sleeping-sickness.

A comparison has since been made between 50 cases having general glandular enlargement without visible cause and 50 with little or no enlargement. Of the former series 20, and of the latter 34, being selected cases, are not included in Table II., and it may be mentioned that in selecting it was more difficult to find cases with no glandular enlargement than the reverse. The resulting figures are not so striking as in the case of the prisoners, but are still suggestive. In the first series, 46, or 92 per cent., were found to have filariæ and nine complained of itching or bad scratch-marks while of the latter, 32, or 64 per cent., showed filariæ and none itching or scratch-marks. Against these, however, must be placed the large number of sleeping-sickness cases in Kavirondo who were examined with negative results as to *F. perstans*, and the majority of whom had glandular swelling.

Should further enquiry show a connection between the enlarged glands and other prominent symptoms of sleeping-sickness and infection with *F. perstans*, the hypothesis might become tenable that sleeping-sickness itself (that is, the lethargy and muscular weakness resulting from brain-inflammation) may be but one, a deadly, but by no means necessary one, in a group of phenomena due to a filariasis in which the parasite is *Filaria perstans*.

With regard to the examination of blood films, fuchsin was the stain almost always employed, after the method recommended in Manson's "Tropical Diseases." To save time a rather stronger solution was used, so as to stain in about twenty minutes, and it was found that this washed out the hæmoglobin effectually. It was also noticed that the worms are easily seen in dried films by merely washing out the hæmoglobin, without staining.

After counting the number of filariæ in many

slides, no relation could be made out as to the severity or stage of sleeping-sickness, nor was there any decided difference in this respect between sleeping-sickness and other cases. The largest number seen in one field (half-inch, Baker) in sleeping-sickness was 13, and the largest number in other cases was nine.

The results as to filaria in Table II. refer, as a rule, to one examination only. In 11 cases of sleeping-sickness in which the first result was negative and in which re-examination was possible, the filaria was found in five; in four at the second, and in one at the third attempt. Of the remaining six cases in which the worm was not found, one was examined 16 times; three, four times; one, three times, and one twice.

No other filaria but *perstans* was found during the present investigations, though several cases of elephantiasis were seen, but the *perstans* embryos seemed sometimes to vary a little in size and manner of staining, even in the same slide. Taking extremes, it seemed possible to distinguish two forms or phases, as illustrated in fig. 1. The difference in size, as measured by the eye, appeared to include all dimensions of the worm, and not to be accounted for by thickening from contraction in length. In a lightly-stained film the larger form is brightly refractile and stains faintly, while the smaller is less refractile and stains almost as deeply as the white blood-corpuscles. In a deeply-stained specimen the larger stains in its axis only, or chiefly, while the smaller stains throughout. The larger is the less usual form. It is met with sometimes alone but generally along with the smaller, and it has occurred in all neighbourhoods and among all classes examined. No explanation is offered of these phenomena, which may be quite accidental, but they are recorded merely as of interest and for further inquiry.

Mosquitoes, &c.—Specimens have been collected where possible from all parts of the epidemic area visited, for examination by experts. For the same purpose, in addition, as many species as were available have been fed on cases of sleeping-sickness having demonstrable *F. perstans*. Also some specimens similarly fed have been examined microscopically, with the following results:—

A. costalis, two examined, second and third day after feeding. Filariae found only in stomach, where some remained alive up to forty-eight hours after feeding.

A. funestus, four examined first to sixth day, no filariae seen.

A. paludis (Theobald), one examined third day, no filariae seen.

Culex (large, yellow and black), three examined second, seventh and ninth days, with results same as in *A. costalis*.

Culex (small brown), one examined fourth day, no filariae seen.

Panoplitcs (*Africanus* of Theobald?), nine examined. In three cases, examined third day, one or two filariae found in teasing of thorax. In one of these, which had died about eleven hours before dissection, a filaria was seen living and actually

motile. Living filariae were found in the stomach up to forty-eight hours after feeding. In those examined after third day no filariae were seen.

Stegomyia fasciata (Theobald), two examined third day. Living filariae in stomach up to forty-eight hours. One filaria found in teasing of thorax which appeared to show degenerative changes.

S. sugens (Theobald), two examined, results same as in *A. costalis*.

Other blood-insects examined were fleas and lice taken from clothing of sleeping-sickness patients, flies resembling house-flies, fed on filarious blood, and a small dipterous insect, very like a house-fly in shape, called by the natives "bwa," which, however, could not be got to feed in captivity and appears to have a limited distribution. In all except the "bwa," filariae were occasionally found in the stomach.

The results of the above examinations were, therefore, practically negative. No developmental changes in the filaria were observed, nor was it seen actually within the thoracic muscular tissue. And though the fact that several were found in teasings of the thorax seems to show that the worm has a certain power of migration in some species, its long stay in the stomach in most cases would count against the probability of its migration for developmental purposes to other parts of the mosquito. However, the prolonged vitality of the filaria seen in one Panoplitcs might be thought to cast a certain amount of suspicion on this species, especially since *P. Africanus* has been shown by Dr. Daniels to be filaria-bearing in the case of *F. Bancrofti*. Owing to the small size of the worm it is more difficult to detect in teased muscular tissue than *F. nocturna*, and, unfortunately, the negative results obtained cannot be said, with the apparatus at command, to be in any way conclusive.

The Panoplitcs of which specimens were examined, was the commonest mosquito in the epidemic area at the time of the expedition (in the dry season). It resembles Prof. Theobald's descriptions of both *P. Africanus* and *P. uniformis*, but does not seem to be quite identical with either. It differs noticeably in the colouring of the proboscis, which more nearly resembles that of *P. uniformis*, varying from yellow to brown at the base, and also in the markings of the legs, the femoral markings apparently differing from both species. These vary from fine white bands on a nearly black ground, to irregular ochraceous marks on brown, the mark nearest the apex being nearly always white and more or less of a band. The last two metatarsi, both of the fore and middle pair of legs, seem to be always unbanded, and the side spot seems to be always absent or nearly absent from the middle segment of the abdomen. The eyes are green in colour during life. On the whole the resemblance must be very close to *P. Africanus*, of which it is most likely a variety. However, the species, one or more, will be determined by experts. The larvæ of this mosquito have not been identified, but from its prevalence during the dry season it probably breeds in native waterpots or in pools on the retreating margins of the lake, swamps, &c. The deep foot-

prints of hippopotami in the latter situations form innumerable potential breeding-places, in which larvæ of *Culices* and of *Stegomyia fasciata* have been found by the writer.

An interesting point about these *Panoplites* is that they were found to be infested in considerable numbers with a minute parasite resembling a tick. In some places quite 50 per cent. were attacked, and as many as nine parasites have been found on one mosquito. The same, or a very similar parasite, was also found in two instances on *A. paludis* (Theobald).

The acarus itself (see drawing, fig. 2) is sometimes whitish or grey, but generally orange red, the colour depending probably on the food of its host, as on a gorged mosquito it is always red. It seems to attach itself by preference to the junctions of the thorax and abdomen or of the thorax and head, but also frequently along the lines of abdominal segmentation. It appears to affect the health of its host, which is generally sluggish and does not live many days in captivity. The acarus taken from the bodies of filaria-fed mosquitoes was examined for *F. perstans* with negative results.

PLAGUE.

PREVALENCE OF THE DISEASE.

INDIA.—During the two weeks ending August 30th and September 6th, deaths from plague in India numbered 4,862 and 5,550 respectively. The chief increases were in the Bombay Presidency (districts), Madras Presidency, the United Provinces and Mysore.

EGYPT.—During the weeks ending September 7th and 14th, the number of fresh cases of plague in Egypt amounted to 5 and 3, and the deaths from the disease to 2 and 1. All the cases occurred in Alexandria.

CAPE OF GOOD HOPE.—No cases of plague in Cape Colony since the middle of August.

HONG KONG.—On September 18th the report from Hong Kong was "No cases of plague for ten days."

MAURITIUS.—During the week ending September 25th, 5 fresh cases of plague and 4 deaths from the disease occurred in Mauritius.

CHOLERA.

EGYPT.—During the weeks ending September 1st, 8th and 15th, the cases of cholera in Egypt numbered 3,875, 7,758, and 9,466 respectively; and the deaths from the disease during these weeks were returned as 2,890, 6,332 and 8,278. On September 15th, 1,557 towns, villages and Ezbehs were infected throughout Upper and Lower Egypt.

MANCHURIA.—In the province of Mukden, between July 3rd and 14th, 757 cases of cholera were recorded, of which number 644 died, 81 being Russians and 363 Chinese. Between July 15th and 23rd, 106 Russians and 276 Chinese died of cholera.

Business Notices.

1.—The address of the JOURNAL OF TROPICAL MEDICINE to Messrs. BALE, SONS & DANIELSSON, Ltd., 83-89, Great Titchfield Street, London, W.

2.—All literary communications should be addressed to the Editors.

3.—All business communications and payments should be sent to P. Falcke, Secretary to the JOURNAL OF TROPICAL MEDICINE. Cheques to be crossed London and South Western Bank, Great Portland Street Branch, London, W.

4.—The Subscription, which is **Eighteen Shillings per annum**, may commence at any time, and is payable in advance.

5.—Change of address should be promptly notified.

6.—Non-receipt of copies of the JOURNAL should be notified to the Secretary.

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THE

Journal of Tropical Medicine

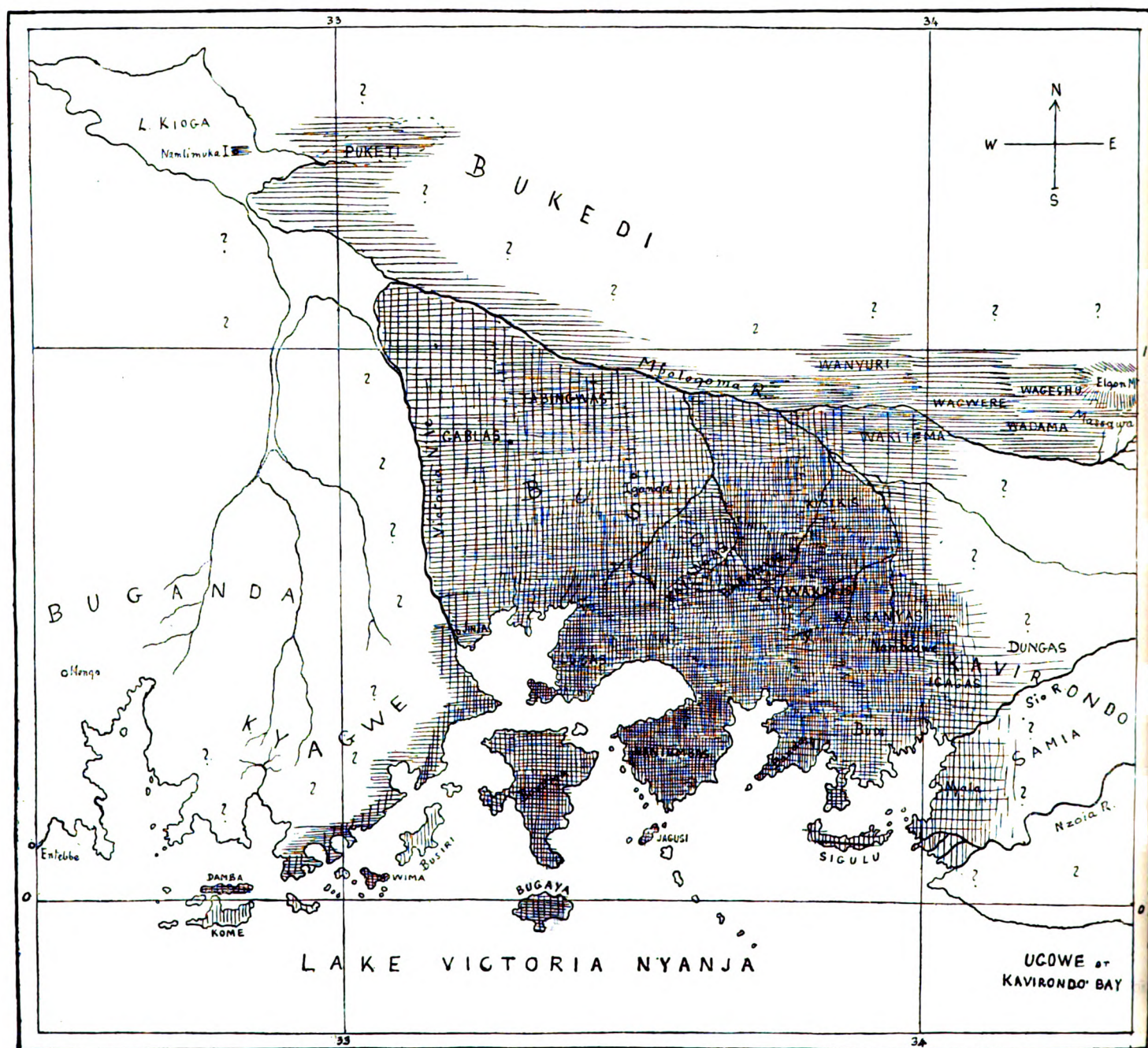
OCTOBER 1, 1902.

PRIZE ESSAYS ON SUBJECTS CONNECTED WITH TROPICAL DISEASES.

We have the privilege of again offering three prizes for competition in connection with this Journal. The subjects chosen include one which is surgical for the most part, one which appertains to medicine, and a third which is devoted to sanitation and public health work.

We are encouraged to repeat these prizes owing to the wide-spread interest created by the last competition and by the excellent papers sent in by several competitors. The three public-spirited friends, to whom we were indebted for the previous prizes, have again come forward and most generously granted us opportunity to stimulate work in connection with tropical diseases.

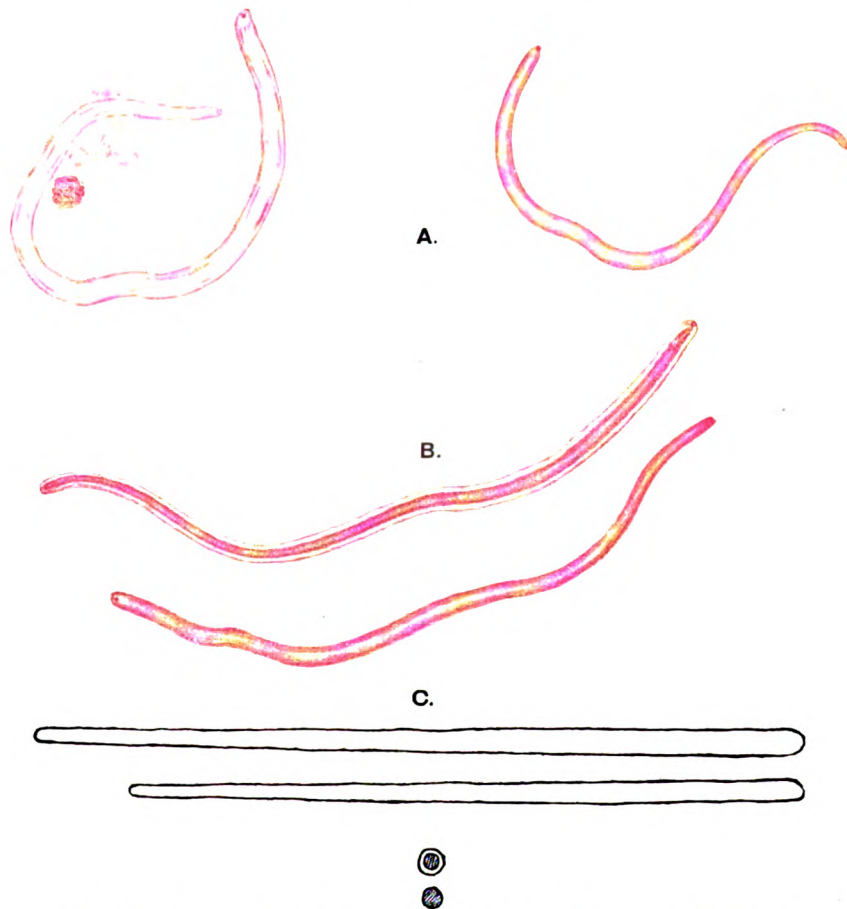
The prizes are open for competition practically to the world, as the languages in which the competitors are allowed to write are, one or other of



MAP-DIAGRAM OF BUSOGA AND NORTH COAST OF LAKE VICTORIA.

The vertical shading indicates the distribution of Sleeping-sickness, and horizontal shading indicates F. Perstans. Intensity of shading indicates high percentage and vice versa. The places marked north of Mpologoma River are those from which cases have been examined.

Fig. I.—FILARIA PERSTANS.



A.—From same slide, lightly stained.

B.—From same slide, deeply stained.

C.—To show apparent relative size.

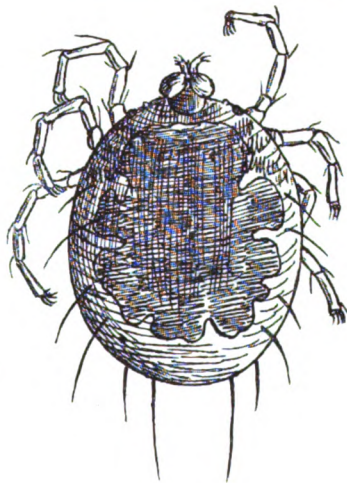


Fig. II.—TICK-LIKE PARASITE
found on *Panoplit*es and on *Anopheles Paludis* (?)
(Theobald).

A.—Under-side of head.

B.—Natural size of parasite.



A

B

them, known to almost all scientific men of any marked standing. We are sorry to exclude the Russian, Greek, and Arabic languages, and especially regrettable is it that Dutch and Portuguese, in which languages so many valuable communications have been made lately, should not be included. The difficulty, however, of obtaining accurate translations of scientific writings of modern medical literature is so great that we hesitate to extend the list of languages mentioned, namely English, French, German, Italian and Spanish.

The cosmopolitan nature of these competitions is evident when the nationality of the recent prize-winners is considered. The Sivewright prize was awarded to Dr. Attilio Caccini, Assistant Physician, Hospital of Santo Spirito in Sassia, Rome; and the Belilios prize to Dr. Bruno Galli-Valerio, Professor in the University of Lausanne. Italy and Switzerland claimed the prizes, and whilst congratulating the winners of these prizes most heartily, it is not a little surprising to find Britain, Holland, France and Germany, with their enormous colonial possessions, failing before Switzerland in a competition in which tropical experience is presumably required. Dr. Bruno Galli-Valerio, of Lausanne, however, dealt with a subject which threatens to spread beyond the tropics. His excellent paper on plague in its relation to the rat shows how a new and rare disease, to keenly scientific minds, is especially interesting. In the tropics the ailments daily met with in the routine of practice are apt to be regarded as the stereotyped setting of every-day life, and the very plethora of opportunity tends to engender neglect of the fact that every European medical man in tropical practice is as yet but part of the advanced guard, a very scout in fact, in quest of knowledge in regions unknown to science. Were these facts kept fixedly before one whilst in tropical practice, a continued interest and stimulating zeal would be the result, rendering the daily round of practice more attractive and enjoyable, and the recording of facts and figures more usual.

Records of experience in tropical practice are not likely to become stale for many years to come.

The medical man who will furnish a list of the cases he has to treat in the course of the year is rendering a valuable service to medicine. The fact that so many cases of gonorrhœa, syphilis, small-pox, bronchitis, &c., &c., or such other every-day ailments, came under his care is a valuable addition to the geographical distribution of disease, and of considerable scientific importance for many other reasons. Every medical man can do this, and we shall have great pleasure in publishing them. Men in practice think that, as they have no time to investigate a subject bacteriologically, their written communications are worthless. There can be no greater mistake, as we have just attempted to show.

THE SIVEWRIGHT PRIZE.

The subject chosen for this competition is a most important one. Diseases of the rectum and anus are a constant source of illness in warm countries. Pruritus ani, fruitful of much discomfort and arising from and complicating many conditions and constitutional ailments, opens a wide field of study in itself. Anal fissure, painful tropical rectal ulcer, post-dysenteric ulcers, &c., in addition to the liability to hæmorrhoids, fistulæ, strictures, &c., in the tropics all come within the scope of this article. The methods of surgical treatment recommended for these conditions is of the first importance, and should form a prominent part in an essay on this subject.

THE BELILIOS PRIZE.

The sanitary requirements of tropical cities is still an open question. The question of the disposal of sewage, whether by the bucket system, by the cesspool system, by the separate system, &c., can only be dealt with by medical men and medical officers of health who have actual experience of tropical work. Experts acquainted only with sanitation in Europe, unless they study the climatic and geographical necessities of a particular area on the spot, are unable to give advice which can be of practical value. The sewerage suitable for the dry season is unsuitable for the wet, and the advisability of a separate storm water channel is still in abeyance. Moreover,

a city on a plain and another on a hill-side or high plateau demand that local surroundings be taken into account. The habits of the natives, their intelligence, and their power of acquiring European notions, have all to be thought of before advising on a question of the kind. We hope the military and naval medical officers may be induced to give their experience in this matter, and that the medical officers of health quartered in various tropical and subtropical towns in British, American, French, German, Dutch and Portuguese colonies may add to our knowledge on the subject.

THE LADY MACGREGOR PRIZE.

The prophylaxis of typhoid by inoculation is a subject of universal interest. Although personal observation on anti-typhoid inoculation is no doubt limited, the literature of the subject is open to every one; and an unbiassed opinion of the usefulness of the treatment may, perhaps, be better arrived at by one who studies the subject as a whole and at a distance, than by one who merely reports what has come under his own observation.

It will be observed that unless intimation is given before February 1st, 1903, that a particular prize is to be competed for, the subject will be withdrawn and another substituted for competition. It therefore behoves intending candidates to announce their intention of competing at an early date.

NOTICE.

LONDON SCHOOL OF TROPICAL MEDICINE.

CERTIFICATES NOW READY FOR DISTRIBUTION.

THE certificates granted to students who have completed a course of study at the London School of Tropical Medicine are now ready, and any student of the School who is entitled to the certificate should apply to the Secretary of the School, Albert Docks, London, E.

DYSENTERY.

R Potass. et sod. tartrat. sat. sol. ʒii.
Tr. camph. co. ʒss.
Syr. rhei q. s. ad ʒiv.
M. S. : ʒii. every hour, or until two or three evacuations are produced.

THE PROPHYLAXIS AND TREATMENT OF BERI-BERI.

By PATRICK MANSON, C.M.G., F.R.S., LL.D.

Medical Adviser, Colonial Office, and to the Crown Agents of the Colonies.

Physician Seamen's Hospital Society.

IN discussing the etiology of beri-beri it is of importance that from the outset we should be in agreement as to what the word beri-beri indicates. We know for a fact that the term has been applied to ankylostomiasis, to epidemic dropsy, to sleeping-sickness, and to a variety of other diseases; and I am not quite sure that even at the present day it is always correctly employed. Indeed it is doubtful, and until we know how to isolate and recognise the specific cause, it will continue to be doubtful if we can diagnose this disease correctly in every instance. I am sometimes tempted to think that even the most experienced tropical practitioners include several specifically distinct forms of neuritis under this one term beri-beri.

The history of malaria should be a warning to us in this matter. Until Laveran taught us to recognise the cause—the germ—of malaria, and thereby enabled us to accurately separate off and define the malarial group, it was often hard to say whether certain tropical fevers were malarial or not malarial. Certain it is that previously many types of fever which we now know were non-malarial were regarded and described as malarial. In the pre-Laveran age all we had to go on, both in classification and in diagnosis, were the ordinary clinical signs and symptoms, and we now know how misleading these have proved to be. In this respect, as it was with malaria twenty years ago, so is it at the present day in regard to beri-beri. Until we have discovered the germ, virus, physio-pathological condition, or whatever the *vera causa* of beri-beri may be, and until we know how to recognise this in our cases, we are sure to be more or less at sea in our definitions, our classifications, descriptions, and diagnoses.

Clinically beri-beri is a multiple peripheral neuritis. From the etiological standpoint, in the tropics as elsewhere, there are many kinds of peripheral neuritis. But, though etiologically distinct, all kinds of peripheral neuritis have necessarily much in common, clinically. Peripheral neuritis is a symptom, or sequela, rather than a disease. Hence a difficulty in differentiating them etiologically, that is, in classifying them scientifically, and in diagnosing morbid causes or diseases this condition belongs to.

Undoubtedly in the tropics there are cases of peripheral neuritis arising from alcohol, from ptomaines of different kinds, from minerals such as tin and arsenic, and from organic poisons developed in the body in the course of specific infections.

In beri-beri districts it must often happen that such cases are regarded as beri-beri. Indeed, it has lately been suggested that many, if not all, cases reputed to be beri-beri are really arsenical poisoning, and the fact that traces of arsenic have been discovered in the hair of such patients has been adduced in support of this hypothesis. I would not deny that arsenical neuritis is uncommon in the tropics, but I feel quite certain that what is generally regarded as beri-beri has nothing to do with arsenic, and is not arsenical neuritis. I

Journal of Tropical Medicine.

PRIZE ESSAYS ON SUBJECTS CONNECTED WITH TROPICAL DISEASES.

1.—A prize of the value of **£10**, entitled the

SIVEWRIGHT PRIZE,

PRESENTED BY THE

HON. SIR JAMES SIVEWRIGHT, K.C.M.G., LL.D.,

FOR THE BEST ARTICLE ON

"The nature and treatment of diseases, exclusive of acute dysentery, affecting the lower part of the large intestine occurring in warm climates."

2.—A prize of the value of **£10**, entitled the

BELILIOS PRIZE,

PRESENTED BY THE

HON. E. R. BELILIOS, C.M.G.,

FOR THE BEST ARTICLE ON

"The system, of drainage and sewerage (domestic and municipal) best suited for tropical climates."

3.—A prize of the value of **£10**, entitled the

LADY MACGREGOR PRIZE,

PRESENTED BY

LADY MACGREGOR,

FOR THE BEST ARTICLE ON

"A critical examination of the practical value of anti-typhoid inoculation."

CONDITIONS.

An intending Competitor must send in his name, address, and the title of the prize to be competed for, to the Editors, "The Journal of Tropical Medicine," care of Messrs. Bale, Sons and Danielsson, 83-89, Great Titchfield Street, London, W., before February 1st, 1903.

The papers to be sent to the Editors of "The Journal of Tropical Medicine," 83-89, Great Titchfield Street, London, W., by May 1st, 1903.

All papers sent in become the property of the Journal, and will be published as the Editors decide.

The competition is open to qualified medical practitioners of all denominations and every nationality.

The papers may be written in English, French, German, Italian or Spanish.

The names of the prize-winners will be announced in July, 1903, in the public press and in the medical journals.

The judges are :—

Surgeon-General ROE HOOPER, C.S.I., President Medical Board, India Office.

Colonel KENNETH MACLEOD, LL.D., Professor of Clinical and Military Medicine, Staff College, London.

PATRICK MANSON, C.M.G., F.R.S., LL.D., Medical Adviser, Colonial Office and Crown Agents of Colonies.

have no doubt arsenic may be frequently discovered in the hair of beri-berics, but, considering the habits of the natives of some of the countries in which beri-beri occurs, this is only what might be expected. The Chinese, for example, frequently use this mineral in the arts, sometimes in agriculture, and very often in their tobacco. Dr. Preston Maxwell lately sent me samples of Chinese tobacco, to which arsenic had been intentionally added with the object of giving it a peculiar and much relished garlicky flavour. This is quite a common custom in China. Habitual consumers of such tobaccos, beri-berics and non-beri-berics alike, would certainly show traces of the mineral in their epithelial tissues, when these were chemically tested. This does not prove that beri-beri is arsenic. I am not aware that any correspondence in the distribution of beri-beri and in the prevalence of arsenic using customs, has been shown to exist. Indeed, in the case of arsenic flavoured tobacco, the opposite is often the case, for beri-beri is specially prevalent in such places as gaols and schools, institutions in which tobacco smoking is not permitted. Further, as an argument against arsenic being the cause of beri-beri, I would mention that it is by no means uncommon to exhibit arsenic medicinally in the later stages of the disease. I have never seen or heard of an aggravation of the symptoms by this practice. Were arsenic the cause of beri-beri, such a practice would surely be hurtful. There are many other arguments, clinical and epidemiological, which might be brought forward to show that arsenical neuritis and beri-beric neuritis are distinctly different conditions.

There is, however, one form of endemic neuritis prevalent in certain parts of the tropics which, judging by its peculiar symptoms, might answer to chronic arsenical poisoning. I refer to the neuritis described by Dr. Strachan as being so prevalent in Jamaica, and probably elsewhere in the West Indies, and which is characterised by trophic skin lesions, such as are common in arsenical neuritis and implication of the higher centres—symptoms in my experience very rare, if not unknown, in beri-beric neuritis.

We often hear of malarial neuritis, and, sometimes, of small epidemics, even of what is described as malarial neuritis. I feel convinced that the vast majority of these cases and epidemics were really beri-beri. I believe there is such a thing as malarial neuritis, but, judging by my experience, it is an exceedingly rare condition, and never occurs as an epidemic. It is not enough that a neuritis occurs in a malarious district, or that it follows close on a malarial attack, to warrant the conclusion that such neuritis is malarial. Men break their legs sometimes in malarial districts, and, doubtless, even during or immediately after malarial attacks; but this does not prove that the fracture was malarial. So in regard to some recorded instance of so-called malarial neuritis. At present there is in the Seamen's Hospital, Albert Docks, a man suffering from well-marked beri-beri, who at the same time is the subject of a concurrent malarial infection.

The patient is a Danish seaman, sixteen years of age. Last December his ship was on the Congo, and there, on the 25th of the month, he got his first attack of fever. It was tertian in type, and was promptly checked by quinine. The ship sailed for Santos, in

the Brazils, on January 14th. On the voyage he had a recurrence of his fever, which was again checked by quinine. The ship left Santos for Falmouth in March. Three weeks out his legs began to swell. Presently the œdema extended to the trunk, and he suffered much from breathlessness and palpitations. During the last fortnight of the voyage he was unable to walk. On arrival in Falmouth he went to hospital, where his case was regarded and treated as one of beri-beri. He remained in Falmouth for a month and then came on to London. On admission to the Seamen's Hospital, on July 9th, his temperature was normal, but it was noted that his heart was extremely irritable. He had marked foot-drop, hyperæsthesia of the calf, thenar and hypothenar muscles, weakened hand grasp, absent knee-jerks, numbness of shins, high stepping gait, and other signs of peripheral neuritis. The œdema had disappeared. His spleen and liver were markedly enlarged. On the following day his temperature rose to 101.4, and on microscopic examination malignant tertian parasites—fever forms—were found in his blood. After a day or two he was put on euquinine. His fever quickly yielded, but by July 17th crescents had begun to appear in the peripheral circulation.

There can be no question but that this lad was the subject of a malarial infection and concurrent beri-beri. The blood examination proved the malaria, and the character of the symptoms, together with the fact, which he mentioned, that one of his mates had died on the voyage with dropsical and paretic symptoms exactly like his own, proved the beri-beri. As malarial neuritis is a rare disease, it is in the highest degree improbable that two cases of a very rare condition should thus occur simultaneously in one ship's company.

In the same ward with this patient there is another malarial case with pronounced tubercular disease of the lungs. The usual physical signs and tubercle bacilli in the sputum are present. The neuritis in the one case is no more the result of the concurrent malarial infection than is the tuberculosis in the other.

The diagnosis of the different forms of peripheral neuritis is often from the clinical symptoms alone extremely difficult, if not impossible. If I were asked what clinical points I would rely on in making a diagnosis as between beri-beric and malarial neuritis, I would say, for the former, marked proneness to cardiac implication; for the latter, impairment of memory. We have as an out-patient at the Seamen's Hospital just now a man who, last October and November, had severe and well-marked malarial fever when in Northern Nigeria. He was treated with quinine and got over the fever but immediately developed intense neuritis which, in a short time, culminated in complete paresis of the lower limbs, weakness of the upper extremities, and a high degree of hyperæsthesia of the calf muscles. At one time he was quite unable to walk. He is now slowly recovering and can walk a mile or two without much difficulty. I have seen a good many similar cases from West Africa and am often puzzled about their diagnosis. One and all, including the patient I have just referred to, have complained of loss of memory—"West Coast memory" it is called in that part of the world, where it is a

well-recognised condition. I have not remarked this loss of memory in beri-beri. It is certainly often a pronounced feature after malaria. I have seen patients in whom this loss of memory has been complete. I am attending just now a gentleman, fifty-one years of age, who, after a short stay on the West Coast, contracted a severe malarial infection. This was in March of this year. Since that time his memory for events occurring during and after fever is a complete blank. He is not very anæmic, but his spleen was a short time ago distinctly enlarged, so that there can be little doubt as to the nature of the fever he suffered from. He can converse quite rationally on matters referring to dates antecedent to his fever, but his memory for recent events is so bad that he cannot tell even what he has had for breakfast five minutes after he has eaten it. I may be wrong, but I think this point of loss of memory is of some diagnostic value in deciding between a malarial and a beri-beric neuritis. It is of less value in diagnosing certain other forms of neuritis from the malarial type, for we know that alcohol and the toxins resulting from a variety of other infections, and diseases producing anæmia, often give rise to the same condition.

Whilst fully recognising its inadequacy and the possibility that it covers forms of neuritis other than beri-beri, I would suggest the following features as a basis on which to found our recognition of that disease as a distinct form of multiple peripheral neuritis, and as more or less distinguishing it from other pathological groups with neuritis as their leading clinical phenomenon: (1) Our ignorance of its cause. (2) Its occurrence as an endemic and epidemic disease. (3) Its proneness to produce cardiac disability and dropsy. (4) Non-implication of the cranial nerves with the exception of the pneumogastric. (5) Non-implication of the intellectual and emotional centres. (6) Rarity or complete absence of trophic skin lesions. (7) High rate of mortality under certain conditions.

I shall take it, but always with the reserve I mention, that a neuritis exhibiting these features is beri-beri.

In considering the etiology of a disease, it is convenient to divide the subject into (1) the immediate cause—germ, toxin, inadequate or improper food, traumatism or whatever it may be; (2) the means by which, or the medium through which, the cause is applied; (3) the circumstances personal to the individual which influence his receptivity and susceptibility; (4) the physical conditions external to the patient, favourable or the reverse, to the application and operation of the cause.

It is a somewhat humiliating fact that, although beri-beri is a disease of first-class importance in the tropics, although it exhibits peculiarities in its epidemiology so striking that they seem to suggest that surely the cause cannot be hard to find, and that although not a few investigators, medical and lay, have diligently set themselves to find this cause, we are about as ignorant of its true nature and of the medium in which it is applied, and of the other etiological circumstances, as was Bontius when he wrote about beri-beri over 250 years ago. Quite recently there may have been some advance, but even these recent advances are more in the direction of

showing what beri-beri is not, rather than in the direction of showing what it is.

I think I shall best provoke useful discussion if I state my own view on the etiology of the disease, giving briefly some of the facts and arguments on which these views are founded and, in the course of this *exposé*, discussing or alluding to other current hypotheses.

Assuming and once more emphasising that I do not lose sight of the fact that it is an assumption that we are dealing with only one form of neuritis in what we call beri-beri, I hold that this neuritis is produced (a) by a toxin, (b) the product of a germ operating in (c) some culture medium (d) located outside the human body. Further, I hold that (e) the said toxin enters the body neither in (f) food, nor in (g) water; and am thereby forced to conclude that it is introduced (h) through the skin, or (i) that it is inhaled.

I may be all wrong in this view; to-morrow some new fact may upset it. But for the present this is the result of my study of other men's work and of my own observations. At all events it is the thesis I propose and am prepared to defend. The various points advanced I shall deal with seriatim, but necessarily briefly. I shall allude in some detail to certain recent, and some of them unpublished, observations, saving time by assuming your acquaintance with the less recent literature of the subject.

(1) *The immediate cause of beri-beri is a toxin.*

The analogy of many, possibly, with the exception of leprosy, of all known forms of peripheral neuritis encourages this view. But besides analogy we have other and direct evidence in support.

It is now generally recognised that the most important measure in the management of a case of beri-beri is removal of the patient from the place in which he sickened. If this be done at a stage of the disease sufficiently early, and, provided the dose of virus has not been overwhelming, in the course of a short time—generally a week or ten days—the symptoms begin to mend, at all events not to advance, and, if the patient survive the first fortnight after removal, he almost invariably recovers. On the other hand, his mates who may have been left in the endemic spot will continue to be ill for months, and many of them will very likely die.

This is our experience at the Seamen's Hospital, Albert Docks. The figures I have already published, and therefore need not repeat them. I prefer to bring forward later and more extensive evidence, collected by one who at the time was committed to no opinion.

Dr. Travers, of the Federated Malay States Medical Service, in a paper which I trust will be published soon, states that the new gaol in Kuala Lumpur—the Pudoeh Gaol, as it is called—was occupied for the first time in January, 1895, the building which has come to be known as the Old Gaol being vacated at the same time. For many years no case of beri-beri had arisen in this Old Gaol. In the month of August following the transference of prisoners beri-beri broke out in the new or Pudoeh Gaol. The mortality ran high. No fewer than thirty-two cases occurred in September, and there was a case mortality of 31.7 per cent. The authorities, remembering its salubrity as

regards beri-beri, on and after October 1st retransferred all the cases of beri-beri in progress at the Pudo Gaol to the Old Gaol, and also all cases subsequently originating there. Immediately the case mortality began to fall; from the 31.7 per cent. in September in the Pudo Gaol, it fell in the Old Gaol in October to 15.57 per cent.; in November to 6.15 per cent.; and in December to only 4.25 per cent.

On this and similar experiences I argue that the cause of beri-beri cannot be a germ living and multiplying in the body of the patient, for, if it were such, the patient when he left the endemic spot would still carry the germ with him, and the disease it produces would continue until immunity had been acquired. You cannot get rid of a scarlet fever infection, or of a small-pox, or of an ague, or of a syphilis, merely by leaving the place where the germ of these was acquired. But you can get rid of a toxin in this way, and if the dose of toxin is not renewed the effects of the initial dose will gradually subside. Stop his drinks, and the subject of alcoholic neuritis after a week or two, if he survives, begins to mend.

Pekelharing and Winkler, who regarded beri-beri as an infection by a bacterium, attempted to get over the difficulty suggested by this fact of the improvement of patients on leaving the endemic area, by assuming that the bacterium is and must be frequently introduced into the body by repeated infections. This is a roundabout way of getting over an obvious objection to their germ infection theory. Their assumption is quite unsupported either by fact or analogy. Moreover, the germ they supposed to be the cause of beri-beri is now quite discredited.

As you are doubtless aware, so-called beri-beri germs have been described as occurring in the blood of beri-beri patients. Very few observers have found the same germ, and many careful observers have failed to find any germ whatever. The latest observations in this direction with which I am acquainted are those by Dr. Arthur Stanley, Health Officer of Shanghai. They appear in the current number of that valuable publication, the *Journal of Hygiene*. Dr. Stanley summarises his work as follows: "Thirty cases (of beri-beri), where the symptoms were well-marked and in stages both before and after loss of knee-jerks, were examined. A band being placed round the arm to distend the veins, the bend of the elbow was sterilised by 10 per cent. lysol in strong alcohol repeatedly rubbed in for half an hour, and then washed with ether. The needle of a sterile all-metal syringe was plunged into the median cephalic vein, and 1 cm. of blood withdrawn. The blood was examined under the microscope directly, and was stained with methylene blue, with a negative result. Tubes of peptone bouillon, gelatin, agar, and blood serum were inoculated with two or three drops of blood in each; deep stabs in glucose-agar were also made. Beyond the adventitious inoculation by *Staphylococcus aureus* and *M. tetragenus* respectively, of two out of 150 tubes inoculated, all remained sterile. Six rabbits were injected simultaneously with 1 cm. of blood from six well-marked cases of beri-beri, but nothing resulted."

I fancy had Pekelharing and Winkler and their followers worked as carefully as Dr. Stanley did, we would have heard less about bacteria in the blood of beri-berics. Observe, Dr. Stanley sometimes worked

with blood from cases before the loss of knee-jerk; that is to say, during the period, were the cause of beri-beri bacterium in the blood, when, presumably, they would be proliferating and in greatest abundance.

I hold, then, that the established fact that beri-berics begin to recover shortly after they leave the endemic area proves that the disease is not an infection by a bacterium or other germ proliferating in the tissues.

(2) *The toxin of beri-beri is produced by a living germ.*

The proof of this lies in the circumstance that the disease can be introduced into virgin country and there spread. That is to say, the hypothetical cause is capable of being transported and of multiplying. Spontaneous multiplication is a property peculiar to living things. Therefore the originating agent of the toxin of beri-beri is a living thing—a germ.

There are several recorded instances of the introduction of beri-beri into virgin country; for example, by the Japanese into Fiji, and by Annamites into New Caledonia.

In the first instance the disease did not spread to the aborigines; it might, therefore, be suggested that there is no valid proof of multiplication in this case. It might be advanced that each Japanese patient had been infected beforehand in Japan, or in the ship that brought them, and that the infection did not manifest pathogenic properties until certain favouring conditions were experienced in Fiji. The objection, I must say, is somewhat far-fetched, but, granted it might apply in this particular instance, it does not apply to the New Caledonian epidemic, for in it the disease spread from the immigrant Annamese to the aborigines. Nor can it apply to the following very telling and, I believe, hitherto unpublished instance, the facts of which have been but recently reported to the Colonial Office by Dr. J. Bolton, Sanitary Warden, Mauritius.

Diégo Garcia (7° S. 72° E.), the southernmost island of the Chagos group, is a dependency of Mauritius. It is a narrow, horse-shoe-shaped sandbank some thirty miles in length. The soil is principally sand overlying coral. Here and there there is a certain amount of vegetable loam. Vegetation is luxuriant; there are a few forest trees, but for the most part the island is occupied by cocoa-nut plantations. The population amounts to 466, divided between two stations, the larger of which contains 326 inhabitants, the smaller 140. The former, the settlement with which we are concerned, is called Pointe de l'Est. The people, who belong to the African, Malagasy, and Indian races, are well-housed, well-fed, and for the most part healthy. Anopheles mosquitoes are absent and there is no indigenous malaria. There are no important endemic diseases unless it be trismus neonatorum and dysentery. Until recently beri-beri was unknown.

On June 27th, 1900, nine Johannese coolies—eight men and one woman—were landed on this island. Seven of them, it was noticed, had sore eyes, sore mouths, sore gums, and swollen feet. One complained of pains in his legs and walked with difficulty. Three days after their arrival two of them—A and B I shall call them—applied for hospital treatment.

A had swollen and painful legs and a distended epigastrium. Nothing very particular occurred to him

till the month of January—that is, seven months after his arrival on the island. He then complained of dyspnoea, palpitations and general œdema, and his pulse was irregular. He died suddenly on the 15th of the same month.

B had pains in, and œdema of, the legs, but no cardiac distress. He recovered in about a fortnight, apparently in consequence of a purgative.

On August 6th, that is, seven weeks after landing, *C*, another of the same batch of Johannese coolies, was sent to hospital with swollen and painful legs. For a time he was treated as an out-patient, but he, too, early in January, feeling worse, applied for admission. His legs were swollen, painful and tender; his epigastrium was distended, he had much cardiac distress and an irregular pulse. He died suddenly on January 12th.

In August, *D* showed similar symptoms, but they gradually disappeared, leaving his legs paralysed.

E also became affected, and along with *D* and the other surviving Johannese, was sent back to Mauritius in June, 1901.

Five, therefore, out of the nine Johannese were attacked with beri-beri, two of them dying with characteristic suddenness.

Up till the month of March, 1901, none of the other and older residents on the island had been attacked; but on the 27th of that month, the hospital assistant was seized with urgent symptoms of beri-beri, and died suddenly on April 8th. On March 25th, a second resident was observed to be affected; he also died suddenly on April 28th. On April 29th, another death occurred, his case dating apparently from March 15th. On May 9th, a fourth was seized and died suddenly on the following day. On May 10th, a fifth, a woman, was attacked; she died on the 22nd. On June 11th, a sixth, the husband of this woman, fell ill and died five days later. On June 15th, their child sickened in the same way, and died on June 23rd. Between the last date and July 1st, when the epidemic ceased abruptly, nine additional cases occurred, but all of them recovered, although at the time of Dr. Bolton's visit, six weeks later, some of them showed well-marked paresis and atrophy of the legs.

Dr. Bolton attributes the spread of the disease to the islanders to germ infection derived from the Johannese immigrants; but he does not explain satisfactorily why it ceased to spread, why the islanders themselves did not acquire infective properties. He does mention however, what to me seem to be two most important facts: (1) The surviving Johannese and, it is to be presumed, their filthy belongings were deported on June 11th; and (2) the huts in which they had resided were burned down. Observe, three weeks after this the epidemic, which, seemed to be gaining strength up to that time, abruptly ceased.

This account of the epidemic of beri-beri in Diégo Garcia, which I have condensed from Dr. Bolton's very lucid narrative, distinctly proves that the toxin of beri-beri can multiply, and therefore that the generator of this toxin must be a living organism—a germ.

(3) *The toxin-producing germ operates in some culture medium. This need not be discussed; the proposition is self-evident.*

(4) *That culture medium is located outside the human body.*

Given that beri-beri is produced by toxin, and that

there is no germ in the bodies of beri-berics (as I have sought to prove), it follows that the culture medium also must be outside the human body.

I would point to the analogy of alcohol and alcoholism by way of illustration. Alcohol, the toxin which gives rise to alcoholic neuritis, is produced during the proliferation of a germ—the yeast plant—in a saccharine solution. We may swallow the germ—the yeast plant—with impunity, and we may swallow the culture medium—the saccharine solution—even with benefit; but the product of the operation of the germ on the culture medium—alcohol—the toxin produced outside the human body, is a poison. So I hold it to be with regard to the germ, culture medium and toxin of beri-beri. What the equivalent of the saccharine solution may be I cannot conjecture, any more than I can point to the germ or to the toxin.

This much, however, I assert:—

(5) *The toxin of beri-beri does not enter the human body in food.*

I had arrived at this conclusion long ago, but only by a process of exclusion and on epidemiological grounds; until recently I was unable to point to any direct or experimental proof. This has now been supplied by Dr. Travers, of Kuala Lumpor, and I have no hesitation in saying that his brief but long-withheld paper is far and away the most important contribution on the etiology of beri-beri extant. True that his results are entirely of a negative character, but they effectually sweep away a mass of crude conjecture and narrow down very much the field for future investigation.

For long it has been conjectured that beri-beri depended on food; either on the nutritive value of the food, or on a pathogenic germ introduced into the patient's body with the food, or on an organic toxic agent contained in the food. As an example of the first type of conjecture I would instance the nitrogen starvation theory, founded principally on the experience of the Japanese navy; of the second, Rost's observations on his rice micrococcus; of the last, Miura's fish poisoning and Braddon's germ—altered rice hypotheses. Many arguments and many facts have been brought forward in support of each of these, but to my way of thinking each and all of them are effectually and permanently disposed of by Dr. Travers' observations.

In Kuala Lumpor, in 1895, there were four large institutions under the care of the Medical Department of the State of Selangor, namely, the District Hospital with 450 beds, a Hospital for Incurables with ninety beds, and a gaol with (as I infer from Dr. Travers' paper, although it is not distinctly stated therein) some 350 prisoners. In none of these institutions, with an aggregate of about 1,000 inmates, had a single case of beri-beri ever originated, nor, for that matter, up to the time of Dr. Travers' report did one originate.

As already mentioned, in January of the year referred to the New, or Pudo, Gaol, situated a mile and a half from the Old Gaol, was occupied. Within six or seven months from the date of its occupation the prisoners in this new gaol were attacked with beri-beri and the disease has stuck to it ever since.

The food supplied to each of the five public institutions mentioned, at all events the rice, was of the same kind and of the same quality. It was obtained from Penang by the same contractor, and in no instance

was it stored in Kuala Lumpor for longer than three weeks. Manifestly, if rice be the cause of beri-beri, in the case of the Pudoah Gaol the change it must have undergone to render it pathogenic must have taken place after it was received at the Pudoah Gaol.

I have already referred to the favourable effect on the sick of a transference from the Pudoah to the Old Gaol, an effect in itself highly suggestive of a pathogenic nidus in the Pudoah Gaol. But a further and more telling experiment was instituted, which not only showed the existence of this nidus in the Pudoah Gaol, but conclusively demonstrated that this nidus was not in the food supply.

On October 21st sixty healthy prisoners were transferred from the Pudoah Gaol to the Old Gaol, and from this date till July, 1896, a large number of prisoners (from 72 to 133) were housed in the Old Gaol. The result is startling. Whereas there occurred during these months in the Pudoah Gaol, in a prison population of from 271 to 337 souls, twenty-one to forty-seven fresh cases of beri-beri monthly, not one case of the disease originated in the Old Gaol. Further, from October 1st to December 14th, 1895, the food for the beri-beri cases and the food for the healthy prisoners now located in the Old Gaol was precisely similar in every respect to the food consumed by the beri-beri stricken population of the Pudoah Gaol.

Not only was it similar in every respect, but it was actually cooked at the Pudoah Gaol in the same vessels and at the same time as that for the Pudoah Gaol inmates, that for the inmates of the Old Gaol being conveyed there twice daily in a hand-cart. After December 14th the rations for the prisoners in the Old Gaol were cooked in that building, raw rations, with the exception of rice, being sent daily from the stores in the Pudoah Gaol. The rice came from the same contractor, and was undoubtedly from the same stock in the case of both gaols, as well as in the case of the three other and beri-beri free institutions already alluded to as being under the charge of the local medical department at Kuala Lumpor.

The inference from this notable and, to my mind, conclusive experiment cannot be avoided. Beri-beri has no direct, if any, connection with food, most certainly not with rice. To have this proved is a great step in advance, and I feel sure that you will agree with me that Dr. Travers is to be congratulated on the excellent use he has made of an exceptional opportunity.

Dr. Travers' paper is directed more particularly against the various hypotheses associating rice with beri-beri. He specifies, however, that "all food" was sent during the time this experiment lasted from the beri-beric to the non-beri-beric gaol. I can imagine some one suggesting that possibly some element in the diet of the Pudoah Gaol prisoners, some element in which resided the cause of beri-beri, was not sent to the Old Gaol. But it is difficult to imagine what, under the circumstances, this element could be, and still more difficult to believe that from 1895 to 1901, the six years during which the Pudoah Gaol has been ravaged by beri-beri, this element has been constantly supplied to the prisoners there, but has never been supplied to the prisoners in the Old Gaol, nor to any of the other three institutions at Kuala Lumpor catered for by the same contractor.

One other point I would remark on. Dr. Travers' experiments were made in 1895. He did not print them till 1901. Depend upon it, had he, in the interval between 1895 and 1901, seen a single flaw in the evidence or other source of fallacy, and no one was in a better position to detect such, he would not have brought forward his work.

From this and other evidence I conclude that the beri-beri toxin is not conveyed in food.

(6) *It is not conveyed in drinking water. This is easily proved. Two institutions placed side by side—as, for example, the male and female prisons in Singapore, with identical piped-water supplies—one, the male, is attacked with beri-beri, the other, the female, is exempt. It is not necessary to labour this point. Proofs are numerous.*

We may conclude, therefore, that it is now apparently proved, so far as negative evidence can prove anything, that the virus of beri-beri is conveyed neither in food nor in water. Apart from the influence of high temperature and moisture in favouring the multiplication of organisms, obviously meteorological conditions have no direct bearing on its production. These cannot be transported, and are not so capricious and limited in the details of their distribution as is beri-beri.

I have shown, I hold, that beri-beri is not caused by a germ operating directly in the human body; therefore beri-beri cannot be passed as an infection directly from person to person. I believe I have shown that beri-beri is the result of a toxin generated by a germ located outside the human body, and I have shown that this toxin is not conveyed in food or in water. We must conclude, therefore, that the virus of the disease is conveyed to man either by the air, or through the skin by contact, or by means of some insect or other animal which inserts it under the skin, or by a combination of some of these ways. So far, unfortunately, we have nothing to show either what the toxin is, nor the germ that produces it is, nor what the precise nidus in which it is produced is, nor the way by which it gains access to the body. Whatever the nidus of the toxin-generating germ may be, both nidus and germ are intimately associated with man; one or both have a wide general distribution, but a very limited particular distribution, and both can exist in ships on the high seas as well as in men's houses on shore. Whoever succeeds in reading this etiological riddle will confer an immense boon on multitudes of our fellow men, for it is reasonable to expect that when a cause, requiring conditions so complicated as those demanded by the toxin-generating germ of beri-beri, is once known, it can readily be controlled if not abolished.

When I set myself to write out these remarks. I intended they should include something about the influence of overcrowding, of weather, of labour, of traumatism, and other circumstances on the etiology of beri-beri; but I find, in dealing with the more immediate cause of the disease, I have already arrogated to myself more than my proper share of the time at our disposal. I trust, however, although in opening this discussion I have said little or nothing on these points, they will not be neglected by the speakers who are to follow me.

The Journal of Tropical Medicine.

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Original Communications.

IS SLEEPING SICKNESS OF THE NEGROES AN INTOXICATION OR AN INFECTION?

By Dr. HANS ZIEMANN.

Naval Staff-Surg., H.M.S. Moltke.

(Translated from the German by P. Falcke.)

As far as I know, no case of sleeping sickness of the negroes has hitherto been described in a German Colony. F. Plehn states that he was once informed of two cases in West Africa by his native servants, which terminated fatally after a short time. He does not, however, mention to which tribe the two negroes in question belonged.

Sleeping sickness of the negroes (known as negro lethargy, Congo sickness, sleeping sickness of West Africa, *maladie du sommeil* *doença de somno*, *rit ansi*, &c.) occurs from Senegal down to Angola, and is manifested by sleepiness which finally deepens into complete stupor, and which, after several months or more, rarely after years, leads to a fatal termination through coma from inanition. We shall presently refer to the clinical symptoms. The disease is also reported from the West Indies and British Guiana in negroes that have been imported from West Africa; according to Ferguson, however, cases also occur in Guiana, in native Indians. In Trinidad, where I stayed over three weeks, as also in Jamaica, St. Thomas and Venezuela, I took great pains to search for cases, but without the slightest success. On the coast of Venezuela, where I became acquainted with nearly every place, the disease at all events occurs very rarely, if at all. This circumstance is remarkable from the fact that Ferguson attributes the disease in Guiana, which is close by, to infection through *anchylostomum*, and I was able to confirm anchylostomiasis as occurring apparently very frequently on the littoral of Venezuela.

In Africa, one is aware that the disease only appears

in certain districts, whereas it is not observed at all in regions near by, and that, moreover, it often appears suddenly. In Cameroon, I have never been able to find the disease amongst the native Duala, though diligently sought for. In fact, the disease does not seem to occur at all in this tribe. It is exceedingly characteristic that no undoubted case of sleeping sickness ever seems to have been observed amongst white people, and, moreover, that children under three years of age are very seldom attacked. This fact will be elucidated below.

During my official term of medical practice in Cameroon, 1899-1900, I heard that the disease, which was known to have raged on the Congo for a considerable period, was also causing great loss of life in North Angola, where entire villages were said to be depopulated by its ravages. I therefore requested Dr. Gleim, formerly Consul and at present physician to the Legation, who was leaving for Loango, to direct his attention to this matter; and I communicated the points of view to him which appeared to me to have an etiological bearing on the question. Dr. Gleim was good enough to convey his first impressions to me in writing, and he published a remarkably interesting report on the disease in *Arch. f. Schiffs und Tropenhygiene*, 1900. I intended myself, at the end of my medical term of office in Cameroon, May, 1900, to go first to the Congo and afterwards to North Angola in order to study the disease, and had already obtained recommendations from the Government to the local authorities, when it was found that the usual steamers, for certain reasons, would be unable to carry me from Victoria to the Congo.

Therefore, instead, the relations of mosquitoes to malaria in Victoria, and in Togo in Upper Guinea, were confirmed.¹

¹ H. Ziemann: *Ueber die Beziehungen der Mosquitos zu den Malaria-Parasiten in Kamerun* (*Deutsche med. Wochenschr.*, 1900, No. 25); "Second Report on Malaria and Mosquitoes on the West Coast of Africa." Lecture before the International Congress in Paris. (*Deutsche med. Wochenschr.*, 1900.)

I had previously succeeded in observing an interesting case of sleeping sickness, which afforded the opportunity for a few experiments. Incidentally, during a medical journey of inspection from Cameroon to Victoria, in Victoria, on February 10th, 1901, I came across the negro, Jamba, suffering from sleeping sickness. The man belonged to the constabulary—a Wey negro from Upper Guinea—and was supposed to be suffering from the disease for three months. The illness set in with sleepiness and headaches increasing in intensity. He has been in Victoria for one year, and, a circumstance that is not without importance, was in the same household with several agriculturists. These persons, besides their wages, receive weekly from the Government a ration of rusk and salted pork; more rarely fish. They have to provide their own vegetables, &c. I observe that the members of each tribe of negroes abroad mostly fraternise, even cooking together.

Jamba had previously, it seemed, been in Batanga for two years. In his native land, since his maturity, he appears to have suffered periodically from mild fever. In Batanga he had seemingly always been healthy, and never had a venereal disease. He had never been homesick, and had never been fond of work—in this respect, therefore, a normal negro—and was not given to more alcoholic excess than any other negro. The data above mentioned are given in literature as the etiology of the disease. He had not had sexual intercourse for a long time, as “he always fell asleep soon,” nor had he erections for a considerable period. Assertions of a negro as to time or to personal matters cannot be relied on unless backed up by the statements of others. Conversation was conducted by means of an interpreter. In Victoria he contracted the skin disease, designated *craw-craw* by the country folks, from which negroes, otherwise quite healthy, often suffer. Jamba states that he ate a great deal of rice in his native land, and that in Batanga he partook of much manioc, or cassada. Through the interpreter, I was also informed that in Wey-land, in Upper Guinea, much manioc was also eaten, and there eaten more frequently raw than in Batanga, where, as far as he knew, it was always cooked. In order to be further observed Jamba was taken back to Cameroon and placed in the hospital for blacks.

Clinical History.—A very powerfully-built, dull-minded, and sleepy-looking Wey-negro, well-nourished, and 20–25 years of age. He answers questions reluctantly, but concisely, muttering them between the teeth. He always seeks for a support when standing, and without support staggering is always apparent. His gait without support is reeling and staggering. Nevertheless, he can walk without assistance, but after fifteen or twenty steps he feels his way along the wall or bedsteads.

The conjunctivæ are slightly injected, his gaze is vacant and expressionless. The skin exhibits that yellowish-brown tint often peculiar to the Weys in contradistinction to the brownish-black colour of the Bantus, but otherwise there are no anomalies of pigment or atrophy such as is the case on the backs of the hands in pellagra. There is slight *craw-craw*, causing considerable itching on the skin of the thighs, on the chest and abdomen, and more particularly on

the inner surface of the thighs. This eruption, however, is said to have been present before the itching from which he now suffers became so severe. The erythema consists of a sort of dermatitis nodosa, which in Cameroon is frequent, particularly in the Bush and Croo negroes from Upper Guinea, but which I have never seen in Europeans. Through the confluence of the small nodules, varying in size from the head of a pin to a hemp-seed, the eruption in Jamba—as frequently happens in such cases—consisted of a few beet-like, hard, flat, protruberant agglomerations, on the periphery of which there were isolated fresh papules. In contradiction to F. Plehn, I failed to transmit the disease in two other cases of dermatitis by transferring scraped material from such agglomerations on to the scarified skin of other negroes.

There were a few vaccination cicatrices on Jamba's left upper arm. The skin was very dry, but otherwise there was no exantham or œdema. The inguinal glands on both sides were partly enlarged to the size of a pigeon's egg, they were fairly soft and not sensitive to pressure. The occipital, cervical and supraclavicular glands were not enlarged.

Temperature 36° C. Pulse 60, regular, fairly strong; the radial pulse was somewhat tense. Respiration regular, 24 to the minute, somewhat superficial breathing, costo-abdominal.

Special Symptoms.—Jamba is undoubtedly somewhat stupefied, and can only be kept awake for a few minutes by steadily staring at him, after which he assumes a lateral position, swathes himself in his quilt and goes to sleep. In other respects no abnormalities in the position of the limbs can be confirmed, no atrophies nor paralyses, not even of the levatores palpebræ superiores. The movements of the eyeballs and of the mouth are quite normal, as are also the movements of the head and limbs, passive as well as active, when he assumes the dorsal position. There is no stiffness of the neck. When standing he soon commences to stagger. Romberg's symptom marked. There is an uncertainty in grasping articles, and when the eyes are closed. There is no actual disturbance of speech. Writing cannot be tested, as there is complete ignorance of the alphabet. He complains of headaches, which occasionally become more severe towards evening, also of giddiness. The sleep is profound and dreamless even in the day. Jamba, like all negroes, is hard to awaken. Tremor and tonic convulsions which are frequently observed in this disease, are not present, neither are there illusions or hallucinations; on the other hand, there is dementia and depression. Sensibility diminished over the entire body (in the initial stage it is frequently not yet decreased). The most delicate touches are not felt at all. Sensations of pain caused by pricks of a needle are readily recognised. The sense of temperature is seemingly diminished. The patient's intelligence being of a low type, no decided result of the proof of the sense of locality and muscular sense can be confirmed. Vision, hearing, smell and taste are seemingly not diminished, or at all events, not to any extent. The fundus oculi, on being examined with the ophthalmoscope, is found to be normal. Patellar reflexes are completely effaced. Abdominal reflex is not present, neither are tendo-achilles nor triceps reflexes. The

reflexes of the cornea and pituitary mucous membrane are maintained. The equally dilated medium-sized pupils react slightly to light and converge. The electrical excitability is found on trial with the faradic current to be diminished, as well as the gross motor power.

The lips and tongue are somewhat dry, the tongue slightly coated; it is protruded straight but somewhat tremulously; there is no malodorous saliva, such as has been graphically described in other cases. The appetite is good, thirst normal. Jamba at first received the usual food of the other patients, which consists mostly of rice, biscuit and salted meat, or fish, and fruit, such as bananas. There was no vomiting. The stools are soft and pappy, yellow, and contain ova of *Ascaris lumbricoides*, no *anchylostomum duodenale*, and are not particularly offensive.¹

The abdomen presents no peculiarities, no sensitiveness to pressure such as has been observed in other cases. The liver and spleen not enlarged. The respiration is uniform. Nothing abnormal in the lungs, heart and the large vessels was apparent. Number of red blood corpuscles in 1 cbmm., 4,321,570. Number of white blood corpuscles in 1 cbmm., 23,500. There is thus some hyperleucocytosis. The eosinophile cells are double the normal. Very rarely there is fine basophile stippling of the red blood cells. In other respects the red blood corpuscles, in size, &c., appear normal. No malaria parasites. Hæmoglobin contents, with the Fleischl apparatus, 58. Embryos of *filaria perstans* in the blood; about one *filaria* embryo in about ten to twelve fields of vision. At the bacteriological examination of the blood taken from the cephalic vein, it was found completely sterile when examined on gelatine and agar-agar. Micturition voluntary and without pain. Quantity of urine in twenty-four hours, 2700. Specific gravity, 1009. Urine yellowish, clear, without albumen, sugar, or other constituents.

Jamba was at once submitted to an expectant treatment; lysol, ichthylol and chrysarobin have no effect on the dermatitis. On February 13th, santonin is tried for three days with castor oil. On February 18th the ascarides have disappeared. The physical condition is the same as usual. The temperature was never above 37°. The temperature mostly fluctuated between 36 and 36.3° C. The embryo of the *filaria perstans* remained unchanged night and day on repeated examinations. Methylene blue 1:0 in capsules, daily administered for ten days, had no effect on the filariæ.

Repeated examination of the blood exhibited the same condition, being always sterile in the bacteriological sense. Morphologically also, it remained unchanged. On February 13th inoculation of two small grey monkeys, two cats, one guinea-pig, and two fowls intramuscularly with 1½ ccm. defibrinated blood taken from Jamba's cephalic vein. The animals remained healthy. There was no development of embryo of the *filaria perstans*, such as has been assumed as a matter of course, on the grounds of

earlier experiments. On February 20th and 28th, March 4th and 12th, the blood of these animals exhibited no peculiarities. On March 28th, the animals were killed. The blood, sterilised and streaked on agar-agar and gelatine, exhibited no bacterial growths. Incubators heated by petroleum were not attainable, but as a rule they do not act well. The temperature of the chamber where the tubes were kept fluctuated between 22-28° C. The organs of these animals exhibited no deviation from the normal. Of course care must be taken not to ascribe too much significance to such negative results from experiments on animals, an error into which Cagigal and Lepierre fell in their experiments when they affirmed that they had obtained positive results and a new bacillus.

February 28, 1901.—Jamba becomes more and more sleepy, has to be awakened for meals, but eats with a good appetite. As soon as the last bit has been swallowed he is again asleep. When stood up he can no longer move forwards, or at most can only take a few wavering steps and then sink down powerless. In the meantime the external condition of nutrition remains good. The complaints of headaches, especially in the evenings, continue; muscular power is diminished, and also the electrical excitability. The replies have now become monosyllabic. The eyes are now only rarely opened for a short time. The mental obtuseness has also undoubtedly increased. During the last days the increasing sensitiveness to cold is remarkable. Jamba carefully wraps himself in several covers. In the meantime the temperature of the body has only once fallen in the evening to 35.8° C., otherwise it varies between 36-36.3°. Filariæ unchanged. They stain excellently according to my method of staining (*Centralb. f. Bakt.*, &c., 1898, No. 25), or by means of hæmotoxylin. The remaining conditions also are unchanged. Number of red blood corpuscles, 3,980,720; number of white blood corpuscles, 22,300. The number of the eosinophile leucocytes average $\frac{1}{3}$ of all the leucocytes. The red blood cells in other respects morphologically present no peculiarities. Hæmoglobin contents 54, according to Fleischl. Treatment with Fowler's solution, commencing with 8 drops, three times daily; internally, strychnine, 0.05, three times daily. At that time I knew nothing of the treatment with testicle extract.

March 1.—Jamba's fellow-countrymen came and averred they knew the disease well, and said that Jamba would now soon die. He must now return to his "country" in order to meet his family. Those who are acquainted with the difficulties of medical practice in Africa, know how jealously certain tribes watch that none of their relations are submitted to an autopsy, and can understand that Jamba, unfortunately, had to be discharged. On March 1st a photograph of the man was taken in front of the Government Hospital. On March 4th he was sent home on the *Wörmann* steamer. The condition of the skin disease unchanged. Later fate unknown.

EPICRISIS.

The picture of disease described above undoubtedly shows us a typical case of sleeping sickness of the negroes. Unfortunately, however, no contribution to the pathological anatomy of sleeping sickness can be

¹ Mense, in the *Arch. f. Schiff- und Tropenhygiene*, 1900, p. 364, in an interesting article on sleeping sickness, describes sour masses of mucus with a fishy smell in the stools of one patient.

added. A meningo-encephalitis has been known to be present a few times, once also an enlargement of the pituitary gland and changes of the medulla oblongata. Attention has also been called to the resemblance of sleeping sickness to Wernicke's acute "poliencephalitis superior." Meanwhile, the entire epidemiological and clinical condition constrains one to the opinion that it is a disease *sui generis*. According to the verbal statements of Dr. Richter, naval staff-surgeon, Dr. Larranga, in Loanda, observed no visible changes in the brain in autopsies. Let us now turn to the

ETIOLOGY.

The theories that the disease is originated by poisons surreptitiously administered, Indian hemp, mental disturbances, sunstroke, scrofula, &c., have been with justice dismissed as untenable and contradictory to the above-mentioned epidemiological fact. They are hypotheses unsupported by experience, advanced without practical knowledge of the conditions. The assertion that the uncivilised negro of West Africa becomes neurotic from emotion must be of rare occurrence in his child-like nature. In the civilised West Indian negroes, such a predisposition would be far more likely, on account of their intensely psychological life. Mental diseases certainly do occur in the negroes of West Africa. As an instance, I myself observed a case of acute religious mania in a black Presbyterian. Nevertheless, actual diseases of the nerves are very rare in West Africa.

The hypotheses as to sleeping sickness are, first of all: (1) *Manson's theory*, this authority being of opinion that *filaria perstans*, when localised in the cerebral vessels, is the cause of the disease. In Cameroon I have so frequently found *filaria perstans* in the blood of perfectly healthy negroes that it would be remarkable if the filariæ did not occasionally set up cerebral symptoms, provided the correctness of Manson's theory is assumed. In the meantime, *sleeping sickness in Cameroon is quite unknown to the Duala*. The embryos of *filaria perstans* in Cameroon, at least as regards the series of cases known to me, are to be regarded as harmless or fairly harmless blood-parasites, which cannot be made to propagate by the artificial inoculation of blood into other persons, and which therefore can only be transmitted by mosquitoes. *I may here mention incidentally as an extraordinary fact that although I have examined the blood of several thousands of whites and blacks, I have never found filariæ in any but negroes.* Dr. Low, who is Dr. Manson's pupil, and who was commissioned by the London School of Tropical Medicine to investigate the disease, told me personally in Trinidad, that he had frequently found filariæ in whites in the West Indies, a new proof as to differences existing in the separate tropical countries. *Moreover, sleeping sickness has often also been found without the embryo of filaria perstans having been present.*

According to this argument, *filaria perstans* should not be denoted as the cause of sleeping sickness, neither should the change of the pineal gland be thus regarded, as this condition has only once been found.

(2) *Anchylostomum duodenale* and *rhabdonema strongyloides* cannot come into the question as the

cause, for the simple reason that they have only been found in a fraction of the cases.

(3) *Fränkel's diplococcus pneumoniae*, which Marchoux¹ endeavoured to bring into etiological connection with sleeping sickness, may be dismissed for the same reason.

(4) *Cagigal and Lepierre's bacillus*² may also be neglected for the same reason.

Then what is the cause?

During my six months' investigatory journey in Italy I was able to study pellagra frequently and in its most various forms, the disease being very widely disseminated in many districts of Italy. This circumstance directed my investigations on sleeping sickness to other points of view. Pellagra is known to be a disease of *intoxication*, attributable to the continuous eating, for long periods, of rotten maize, causing extensive disturbances of the nervous system. It is advisable that before beginning the study of sleeping sickness that the investigator make himself acquainted with pellagra. I hardly believe that one of the authors who has hitherto written on sleeping sickness has had the opportunity of studying pellagra. It is, however, unconditionally necessary for the investigator to be in the position to make comparisons. It is also very desirable that a minute knowledge of lathyrism and atripticism—both of which are diseases of intoxication and lead to nervous disturbances—be acquired for the same object. Beri-beri will be, moreover, mentioned below.

Sleeping sickness is generally regarded as contagious by the negroes, the Wey-negroes—the constabulary—being likewise of this opinion. The saliva that drops from the mouth of the patient into the food-dish common to the family is especially considered infective. We shall see that there is a spark of truth in the observation of these children of Nature. It is not, however, the saliva of the sick that drips into the dish, that appears to be the cause, but the unsuitably prepared contents of the common dish. It is not a *sine quâ non* that every one of those feeding should contract the disease. *Even amongst those of the black races there are individuals with greater, and individuals with less, predisposition to a disease; so it is also in Italy. It is not every person eating bad maize that is attacked with pellagra.* It appears that it is possible for the hereditary predisposition to be acquired. Of course accessory causes, such as bad food, hardships and excitement, combine in playing a part.

There is no *infectious* disease, at least none the exciter of which is known, that is confined to a single race. Certainly some races are attacked more slightly and sooner than others. I am alluding to the particular predisposition of negroes to pneumonia. It is by no chance that the negroes of the West Indies, when attacked by leprosy, acquire the tubercular form in the multiplicity of cases, whereas the imported coolies

¹ Marchoux E.: *Rôle du pneumocoque dans la pathologie et dans la pathogénie de la maladie du sommeil* (Annal de l'Inst. Past., 1899, No. 3).

² Cagigal, A. O., and Lepierre, C.: *A doença do sono e o seu bacillo* (Coimbra medica, 1897, Nos. 30 and 31.) [According to Mense's reference, Archiv. f. Schiffs- u. Tropenhygiene, 1898, p. 110.]

of the East Indies acquire the anæsthetic form, as I was able to observe in Trinidad and Jamaica. Nevertheless, no race is completely spared.

Why, however, does sleeping sickness attack the negro race exclusively, or almost exclusively? Within my knowledge there is no confirmatory evidence of Europeans being attacked by the disease. But even if, later on, a case of sickness of a European were proved, the explanation given by me would still hold good, for, unfortunately, there are some Europeans that live like negroes, or at all events often live with a negress. Even in mulattoes, who live somewhat better than negroes, the disease has only been observed in exceedingly rare cases.

Why, however, are children spared the disease up to two or three years old—this, according to my researches, being the case on the Congo as well as in the Wey-negroes—whereas, for instance, malaria is particularly apt to attack the children? This fact is interestingly elucidated by my mentioning that up to the age of two or even three years the negro children are suckled by their mothers. *The tenderest age, i.e., age of suckling is also entirely or almost entirely free from pellagra.*

Why do entire tracts of land remain free from sleeping sickness whilst others become depopulated, whereas the same diseases,¹ though varying in frequency and intensity are exhibited on the entire west coast of Africa in general? Cameroon, as mentioned, may be designated free from sleeping sickness as seemingly also may Togo, the Togo coast. In the Congo, however, according to Mense, affected regions and free ones are contiguous one to the other.

According to the report of the physician to the legation, Dr. Gleim, the disease in Angola is increasing and appearing in districts where it formerly did not exist. This *might* speak for an infectious disease, but is not necessary, as we shall see below.

There are many districts of West Africa into which small modern migrations of peoples occur, when one tribe pushes into another like a wedge, imparts its customs to the other, and in turn receives new ones from the subjugated tribe. I need only remind the reader of the powerful onward march of the cannibalistic Fan tribe in the Congo district in a north-westerly direction towards the coast, in order to approach the white man to participate in commercial transactions and to break in upon the commercial monopoly of the people on the coast. I was able to observe this interesting comedy during my first stay in West Africa, 1894-95, in the south of Cameroon and in the French Congo. It is necessary to ascertain if these people, when on their wanderings, do not bring practical hints from their native land which their new neighbours have not yet learned to value. I may, moreover, mention that at the present time, West Africa, with all its treasures, being now opened up for agriculture, &c., hundreds and thousands of labourers of various tribes, far from their native land, are employed on the plantations, and that frequently a small tribal museum is formed in the midst of the surrounding black race.

In Africa, as a matter of fact, the conditions necessary to the spread of a disease, even a *disease of intoxication*, are not lacking. A few exceptions have already been mentioned, as, for instance, the apparent absence of leprosy on the coast of Cameroon, while, according to Mense, it is frequent on the Congo.

When, moreover, as has been stated, the disease only appears years after departure from the affected district, the fact must be taken into consideration that at first the happy-go-lucky negro does not notice the very first symptoms at all, and probably may have been suffering from the disease for some time without it having become noticeable. The negro by nature likes sleeping and sleeps much. The tropical practitioner who has to watch at night with negro attendants has reason to know this. Above all, however, the negro from the coast never or very rarely lives alone in another country on the African coast. He always finds fellow-countrymen with whom he nearly always dwells and cooks, *clinging to the customs of his native place*. In nearly every spot, more or less, at least on the west coast, he will find the same *articles of diet*, though, of course, not the same conditions of life. The Duala in Cameroon have different and better foods than the imported labourers, usually blacks, from Upper Guinea.

I doubt very much if cases are known of negroes acquiring the disease several years after leaving their African home in thorough mental and bodily vigour, and living in European capitals in *European style*.

The above data constrain me to the opinion that sleeping sickness is a disease caused by chronic intoxication with a toxic material ingested with the food. The food in question must be a very common one, present everywhere that the negro is, or, at all events, everywhere where the disease exists. On going through the articles of food that may be regarded as occurring *universally*, dried salt water fish and manioc, or cassada, suggest themselves. Of these, however, dried salt water fish is consumed in places where there is no sleeping sickness. Two varieties of manioc are very common, *Manihot Aipü* (Pohl), which is poisonous in the raw state, but is eaten cooked, &c., and *Manihot utillissima* (Pohl), which is not poisonous. *It is a fact that manioc is freely partaken of raw in those countries where sleeping sickness prevails.* It is, moreover, well-known that the preparation of the poisonous manioc varies considerably in the different countries. It is clear that many stages may occur between the raw poisonous manioc and the same root rendered innocuous, corresponding to the various means of preparing it. I know, for instance, that the Duala only eat manioc cooked, *i.e.*, roasted and after having soaked it for a long time.

By chance I have only lately read Dr. Gleim's exhaustive treatise, and I find that he apparently does not accept my manioc theory, as manioc has also been eaten previously in districts where sleeping sickness has not occurred at all. This question must, however, be solved by exhaustive studies on the spot, a method of investigation that Dr. Gleim himself urges, as the statements of negroes are unreliable. At all events, in Cameroon I have always adhered to my manioc theory, and always explained my reasons to medical men and laymen. Dr. Glenn mentions

¹ Leprosy, which, according to Mense, is frequent on the Congo, does not, however, seem to occur on the littoral of Cameroon.

that a Portuguese doctor also told him that manioc root was the cause of the disease. Of course I do not know if that doctor had heard of my opinion, which I had already uttered in 1899, as I can prove, but knowledge travels quickly on the west coast of Africa. *At all events, I maintain that I was the first to ventilate the theory extensively to a large circle.* On this account also, my undertaking to proceed to Angola in May, 1900, by the recommendation of the Government, to study the disease was frustrated at the last moment. A systematic research into the mode of living of the inhabitants of the affected as well as of the non-affected districts is necessary for comparative purposes. The dissemination of *Manihot utilissima* and *Aiphi* in the separate districts, and the manner of use and manner of preparation, should be investigated. The disease that, at the present time, depopulates entire districts of the Congo and Portuguese states may soon disappear by means of the general instruction of the population as to suitable preparation of the root, if, as I believe myself justified in assuming, the cause of the disease consists in the eating of raw or unsuitably prepared manioc.

The question should likewise be investigated as to whether the poison—be it manioc or not—be influenced in intensity by the climatic conditions of the year in question, the telluric influence of the district in question, so that in some years and in some localities the negroes fall ill and in some they do not. As far as I know, the solanin constituents of potatoes may vary. I remember having read an article by Pfuhl, in which the wholesale poisoning of soldiers through the solanin contained in potatoes is described. It may be objected that the European in Africa in out-of-the-way localities also eat manioc in place of potatoes that do not grow in the valleys of Africa. But the cook in the service of the European always serves up his master's vegetables cooked.

It is really surprising that the above train of thought has not been taken up before, and with enthusiasm.

A few incidents in my practice in Cameroon gave renewed opportunity for reopening the question of intoxication in the tropics. Thus, for instance, I succeeded in discovering a plant, *ophirian low cissampeloides* (Planch), Hook, which is used for purpose of poisoning in order to induce *hemoglobinuria* similarly to *Morchella esculenta*. This will be reported on later. On another occasion I was hurriedly called to a plantation where four negroes had died in the morning and five more were seriously ill. There was the fear, which was justified, that many others would be taken ill, and a few new invalids already showed symptoms of an outbreak of illness resembling ptomaine poisoning. The planter was at first inclined to believe that this was caused by poison administered out of revenge by the members of a certain other tribe. The planter had caused two tribes to change their dwellings, and one tribe was very unwilling to quit its old quarters.

A thorough investigation instituted at once led to the conclusion that the disease had undoubtedly been caused by the drinking of water from a water butt, in which the remarkably stupid cook of the patients had allowed a few rotten pieces of papaya and another unknown fruit to float in for several days. The most

thorough evacuation of the stomach and bowels led to the speedy cure of the sick. However, a few stated that for more than a week after they suffered from headache and weakness in the limbs. This short experience has no direct bearing on the subject under discussion, but it shows in which direction in the way of conclusions *per exclusionem* the researches on sleeping sickness should be conducted. The search for new bacilli must, however, always occupy attention in the reasonable investigation of epidemiological facts.

In this manner, men like Robert Koch, Manson and Ross, Laveran, Bignami, King, &c., formed their conclusions in regard to the modern knowledge of malaria, and were able to prove their point by experimental results in animal and human malaria, conducted partly by them and partly by Grassi and others. Whether my assertion that sleeping sickness is really a disease of intoxication in the same sense as pellagra be justified or not, whether my hypothesis that the ingestion of maniol unsuitably prepared causes the disease, be confirmed or not, I consider I am justified in making the following assertion: *In the blood of persons suffering from sleeping sickness there is no exciter of disease that can be cultured by the usual methods so as to be made to develop further.*¹

Other occupations have hitherto retarded the above communication. The journey now again taken to West Africa, will, it is to be hoped, furnish fresh material. Even if the author of this article is on the wrong track, he hopes by means of it to assist in directing renewed attention to the epidemiology of this highly interesting disease. (*Centralblatt f. Bakteriologie, Parasitenkunde u. Infektionskrankheiten.*)

MALARIAL ORIGIN OF ZOSTER.—James MacFarlane Winfield, who in 1895 gave the results of examinations of the blood in eight zoster patients, showing that about 50 per cent. in them were suffering from malarial infection, now reports twenty-five additional cases, all of which had symptoms suggestive of infection, and fourteen of which gave positive evidences of paludism as proved by the presence of the malarial parasite in the blood. The author states that while it is not his intention to assert that this special organism is the only causative agent in the production of this neurocutaneous disorder, zoster (for it is undoubtedly that many different varieties of bacterial intoxication could bring about the same effect), he maintains that the *plasmodium malarie* should be considered one of the causes in a disease that is so etiologically uncertain.—*The New York Medical Journal*, August 2nd, 1902.

¹ This statement may also be applied to beri-beri, as observed in Cameroon. The aspect of the disease called beri-beri by me in Cameroon is similar to that described by Scheube and others, but is apparently much milder, at least in the cases observed by me.

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OCTOBER 15, 1902.

MEDICAL OFFICERS IN BRITISH PROTECTORATES.

WE are from time to time made aware of the fact that for our Protectorates medical officers are engaged and despatched thither by the Foreign Office. We are unfortunately reminded of the fact by virtue of complaints as to pay, leave, rank and pension sent to medical papers by these officers; and it behoves us to enquire into, and to assist as far as it is possible, the conditions under which they serve. Fighting authorities is not the right way for any public officer to remedy an evil; but there are many methods of bringing to light a grievance which are quite consistent with loyal service. Grumbling is again a style of remedy which is not likely to be attended by the best results; yet it behoves every medical man to state what, after practical experience, appear to be the radical defects in a service to which he may happen to belong. It is

usually argued that every man, when appointed, knew the circumstances under which he took up service, and that he has no right to make complaint. Were this principle followed to the letter, we would never hope to see any improvement in our public services. Our Protectorates are at best but tentative in their attempts at Government, and with the development of a new country there must arise modifications in every branch of the public service engaged in administering the affairs of the protected country. In this spirit, therefore, we are willing to deal with the matter, and we are ready to receive and to publish articles concerning the best method of medical administration adapted to our Protectorates. The Foreign Office, we are sure, will willingly modify the present *régime*, if such seems faulty, and were a scheme of entrance and promotion drawn up by officers of experience, there can be no doubt the public service would benefit, and an efficient medical service would be the result.

DISINFECTION BY THE CLAYTON GAS.

A MIXTURE OF SO₂ AND SO₃.

WE have from time to time reported upon the nature and results of this form of disinfection, and we await with interest the publication of careful experiments which have just been made by Professor Calmette in France. As yet we have the barest outline of these experiments, but they are so important and convincing that it may be well to give a short account of them. The principle of the Clayton method of disinfection is simplicity itself. Take, for example, a ship in which plague has occurred. As soon as the vessel is berthed a barge with the apparatus is towed alongside, and without disturbing the cargo the inlet and outlet pipes are introduced, the former reaching to the lowest part of the hold, and the upper just within the upper part of the deck or decks to be disinfected. The gas, a mixture of SO₂ and SO₃, is generated by the apparatus and conducted by the inlet pipe to the lower part of the hold. Its spread through the vessel is encouraged by the suction of the outlet pipe, and after a period varying from an hour or two to several or many hours, according to the bulk of the cargo,

disinfection is complete. Rats, mice and vermin generally are destroyed, and the cargo is undamaged by the exposure. Germs of infectious diseases are held to be destroyed; but it was not until Calmette subjected the whole process to thorough investigation that a scientific basis for this belief could be said to exist.

Professor Calmette's proceedings were as follows:—

"During the last week of September, 1902, Calmette came down from Lille, bringing with him fresh cultures of the plague, cholera and typhoid fever, arranged in the following manner: Strips of flannel were soaked in the cultures; these were placed in tubes about 30 millimetres in diameter, open at both ends, and stopped with cotton as usual; a set of dry and a set of moist cultures in each lot of tubes. Other similar strips were wrapped in two thicknesses of blotting paper, then in a piece of flannel, and round all a doubled piece of what seemed to me to be thick oiled paper—*double papier écolier gommé* they called it—this making a separate sachet or package for each kind of microbe—one set moist, another set dry.

"A series of each, in tubes and in sachets, was placed in the lower hold of the vessel we were fumigating, and another series in the upper 'tween decks, close to the open 'tween deck hatch, the holds being empty. The deck hatch was only partially closed, as one of our pipes led through it.

"Dry and moist test tubes and sachets were kept for each series; these remained on deck out of reach of the gas.

"At 10.55 a.m. they started the generator, the discharge pipe running to the bottom of the hold through one of the ventilators, the aspiration pipe drawing the air from the hatch. At 11.55 a.m. the gas was found to be 7 per cent. as it came from the generator. At 12.10 it was 8 per cent. from generator; 12.30, 10 per cent. from generator; 12.40, 11 per cent. from generator; 12.55, 14½ per cent. from generator; and 1 p.m., 15 per cent. from generator.

"The atmosphere in the square of the hatch at this hour showed only 5 per cent. of gas, but the generator was stopped at 1.10 p.m., two hours and a quarter from the commencement of the work, the space filled being about 70,000 cubic feet. There was a great deal of leakage through the bulkhead into the adjoining compartment in the ship; and it takes, as you

will see, about an hour for the generator to get properly heated up to produce a fair gas. We could have gone on indefinitely at 15 per cent. after reaching that figure.

"Two hours later, at 3.10 p.m., the hold was opened up, and a score of rats were seen lying about; twenty-seven in all were found. At 5 o'clock, without any artificial ventilation, Dr. Calmette's assistant went into the hold and got out the cultures.

"Another operation was carried out in a deck cabin, where a wet and dry set each of tubes and sachets was placed in the upper and lower bunks. Those in the upper bunk were wrapped up in a doubled woollen blanket and a pillow laid on top of them, with the mattress below. The discharge pipe was pushed in through a port, but no pains were taken to make the place air-tight, and a 4-inch scupper for draining the floor of the cabin was left open.

"At 1.33 p.m. we started the generator, which was still partly hot, and at 1.40 p.m. it was producing a 5 per cent. gas. At 1.58 we had a 10 per cent. from the generator and an 8 per cent. gas in the cabin, when we shut the generator down, leaving the cabin closed.

"At 4.10 the atmosphere in the cabin was tested, and only 1½ per cent. gas was found at the level of the lower berth—thanks to the open scupper I fancy—while 2½ per cent. was found on the upper berth. At 4.15 the door was opened and the cultures taken out; altogether they were in the cabin about two hours and three-quarters.

"Dr. Calmette took all the cultures back to Lille with him that evening, and next morning, September 28th, he placed all his strips—those that had been in the gas and those that had not—in meal bouillon and peptonised water. These cultures were placed in a stove at a temperature of 37° C., and examined in twenty-four hours, three days, and seven days.

"The following table shows the results—0 indicating that the tube cultures remained sterile after seven days in the stove, x indicating that microbes developed.

| | | Plague. | | Cholera. | | Typhoid. | |
|-------------------|---------|---------|--------|----------|--------|----------|--------|
| | | Dry. | Moist. | Dry. | Moist. | Dry. | Moist. |
| A. (lower hold) | Tubes | 0 | 0 | 0 | 0 | 0 | 0 |
| | Sachets | 0 | 0 | 0 | 0 | 0 | 0 |
| B. ('tween decks) | Tubes | 0 | 0 | 0 | 0 | 0 | 0 |
| | Sachets | 0 | 0 | 0 | 0 | x | 0 |
| C. (deck cabin) | Tubes | 0 | 0 | 0 | 0 | 0 | 0 |
| | Sachets | 0 | 0 | 0 | 0 | 0 | 0 |
| D. Tests | Tubes | x | x | 0 | x | x | x |

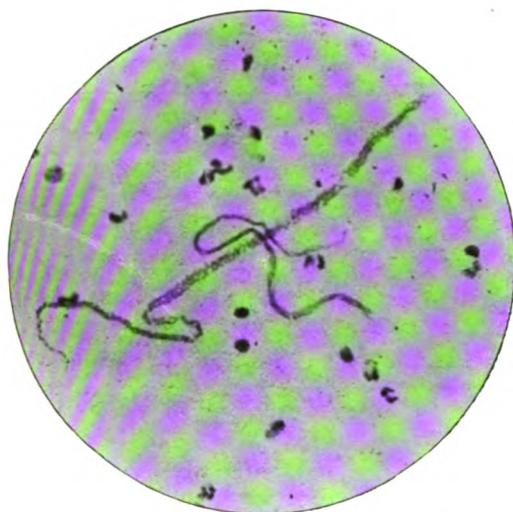


FIG. 1.—*Filaria Nocturna* and *Filaria Perstans* (double infection).

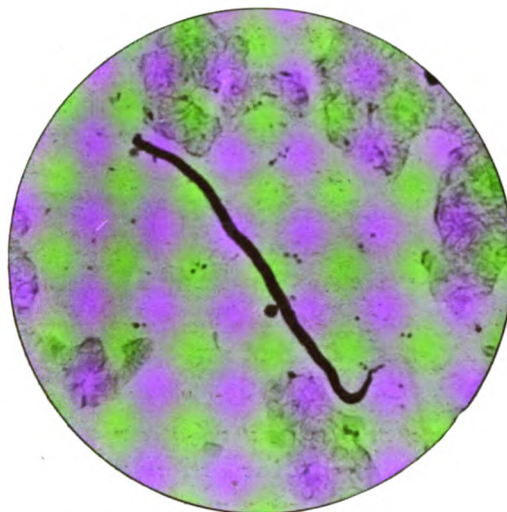


FIG. 2.—*Filaria Diurna*, showing sheath.

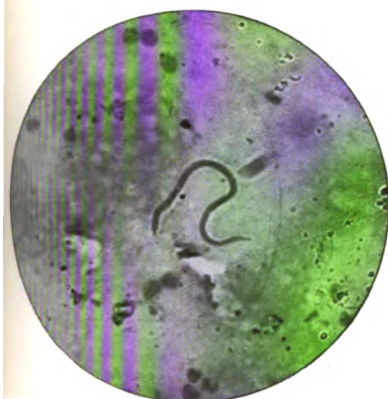


FIG. 3.—*Filaria* (?)

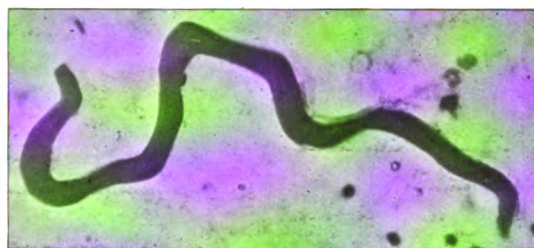


FIG. 5. *Filaria Gigas*.

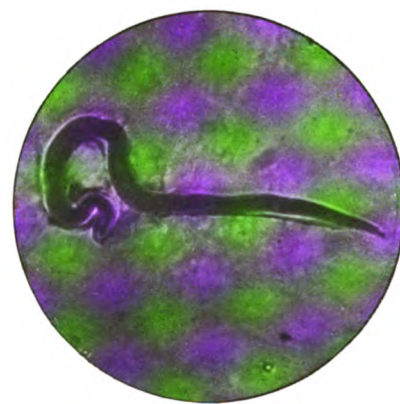


FIG. 4.—*Filaria Gigas*.

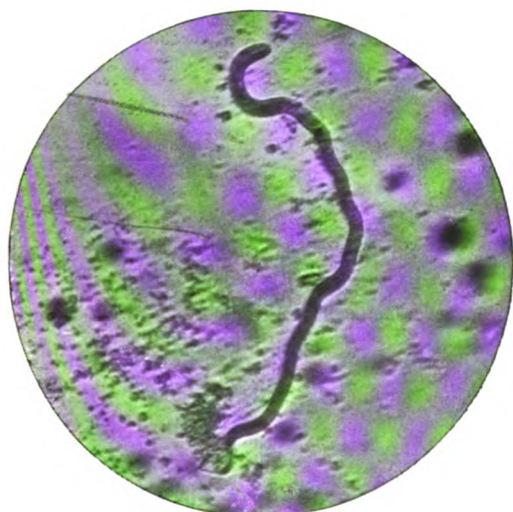


FIG. 6.—*Filaria Volvulus*.

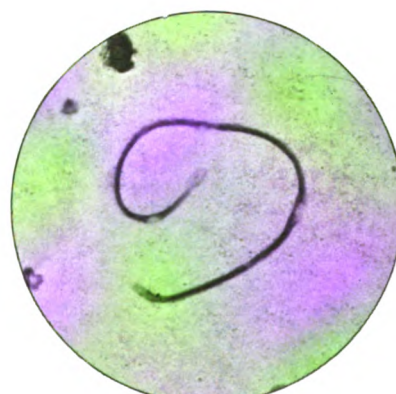


FIG. 7.—*Filaria Loa*.

TO ILLUSTRATE PAPER ON FILARIASIS IN SIERRA LEONE,
By W. B. PROUT, M.B., Ch.M.Edin.

"So that all the tubes with the strips that had been subjected to the Clayton Gas remained sterile except the one that had the dry strip in the sachet placed in the 'tween decks near the hatch.

"All the test cultures that had not been in the gas, except the dry cholera strip, multiplied and flourished abundantly after twenty-four hours in the stove. Dr. Calmette says the cholera microbe in a dry state is very fragile.

"I may tell you that he was very much surprised at the results obtained, and to a great extent so was I. The cultures in the bottom of the hold I expected to see destroyed, but those in the 'tween decks and in the cabin I did not think had been long enough in the gas. Had the operation been prolonged in the hold and 'tween decks for another half-hour, the dry typhoid culture in the sachet would doubtless have been destroyed, as it took longer to develop than the test cultures, and must have suffered to some extent.

"Dr. Calmette has sent an official report to the Minister of the Interior, a copy of which will be published in the *Revue d'Hygiene et Police Sanitaire*. His conclusions are that the gas produced by the apparatus used as is practised in fumigating ships, and of a percentage of 8 per cent., is perfectly efficient for the sterilisation of material tainted by the microbes of plague, cholera and typhoid fever. This process assures the destruction of all rats and insects without damage to goods or vessels, and it ought to be generally adopted for the preservation of the health of crews and passengers.

"All the quarantine stations in France, he says, should be provided with the apparatus as soon as possible, so as to diminish the present long delays in quarantine, and to safeguard the ports against an invasion of plague and cholera, always possible, and at the present moment very threatening.

"He is continuing his experiments on other cultures, and thinks that the gas will prove fatal to the tuberculosis germs."—*Extract from a Letter to the Editor.*

NOTICE.

THE LONDON SCHOOL OF TROPICAL MEDICINE.

LIST OF STUDENTS TO RECEIVE THE
SPECIAL CERTIFICATE.

Staff-Surgeon Percy W. Bassett-Smith, R.N.,
M.R.C.S., L.R.C.P. (1899).

Chas. R. Chichester, B.A., L.R.C.S.I., L.R.C.P.I.,
D.P.H. (1899).

Ernest J. Hynes, M.R.C.S., L.R.C.P., D.P.H.
(1902).

Geo. C. Low, M.B., C.M. Edin. (1900).

Jno. Allen Scotland, L.R.C.P., L.R.C.S. Eng. (1900).

Aaron Sims, M.D., C.M. Aberdeen, D.P.H. (1901).

Jno. O. Summerhayes, M.R.C.S., L.R.C.P. (1902).

W. H. De Wyt, M.B., C.M. Glasgow, D.P.H. Camb.
and London (1901).

The Secretary of the School requests us to state that the above-named are entitled to the special certificates, and that the certificates will be forwarded on application to the Secretary, Seamen's Hospital Society, Greenwich, London.

British Medical Association.

FILARIASIS IN SIERRA LEONE.

By W. T. PROUT, M.B., Ch.M. Edin.

Principal Medical Officer, Sierra Leone.

(With Plate).

THE following notes contain the results of a series of observations on the prevalence and varieties of filaria in Sierra Leone, a subject which, so far as I know, has not hitherto been worked at; and, indeed, with the work on filaria by Dr. Annett and others, published by the Liverpool School of Tropical Medicine, little attention has been paid to this on the West Coast of Africa. My observations tend to show that filariasis is an extremely prevalent condition on the West Coast of Africa, much more so than has hitherto been suspected, and it is not improbable that some of the more obscure diseases with which we have to deal among natives may be connected with this fact.

The principal source from which I obtained preparations of blood was the Colonial Hospital at Freetown, where natives from all parts of the country are admitted, and I was also furnished with a number of slides from the Protectorate through the kindness of some of the district surgeons. In those cases examined at the Colonial Hospital the subjects were suffering from some disease or other, but in the majority of cases from the Protectorate the specimens were from apparently healthy individuals, selected at random from the general community or from members of the Frontier Force stationed in the district. The procedure was in most cases as follows. A thick film of blood was taken in the early part of the day, between 10 and 12 o'clock, and again at night between the same hours. I have not considered it necessary to indicate the number of filaria found in each specimen, as the films varied in size, and so long as the periodicity is ascertained, the actual number of filaria is unimportant, the fact of infection and its nature being the essential features.

I have examined some hundreds of specimens of blood, and of these definite records of 275 have been preserved. In 266 of these, both day and night blood were examined, and in 9 night only. Out of the 266, 57 were found to contain filaria of different kinds, and of the 9, two specimens contained filaria: a total of 57 cases, or 21.4 per cent. of the whole. But an analysis of the figures brings out some interesting results. At Bandajuma, in the Protectorate, 35 per cent. were found to be infected, at Moyamba 38.7

per cent., at Sherbro 22.5 per cent., and at the Colonial Hospital 16.3 per cent. This is brought out in the following table:—

TABLE I.

| | No. Examined | Filaria found in | | | Total | Per Cent. |
|----------------------|--------------|------------------|-------------|------|-------|-----------|
| | | Day Blood | Night Blood | Both | | |
| Bandajuma | 20 | — | 6 | 1 | 7 | 35.0 |
| Moyamba | 31 | — | 10 | 2 | 12 | 38.7 |
| Sherbro | 22 | — | 4 | 1 | 5 | 22.5 |
| Colonial Hospital .. | 202 | 4 | 22 | 9 | 35 | 17.3 |
| Total | 275 | 4 | 42 | 13 | 59 | 21.4 |

It is quite clear, therefore, that filariasis is much more prevalent in the Protectorate than among the cases examined in Freetown. A study of the latter brings out this fact still further. As far as possible I have ascertained the residence of these cases and the length of time they have been in Freetown; 108 were found to have been permanently resident in the city, or had been for lengthened periods. Among these only 6.5 per cent. were found infected, while among those coming from the Protectorate and the outlying districts of the Colony proper no less than 26.1 and 30.3 per cent. were infected.

TABLE II.—Distribution of Filaria among Cases from Colonial Hospital according to Place of Residence.

| | No. Examined | Filaria Found | Per cent. |
|----------------------------------|--------------|---------------|-----------|
| Freetown | 108 | 7 | 6.5 |
| Protectorate | 65 | 17 | 26.1 |
| Distribution of Colony proper .. | 24 | 8 | 30.3 |
| Foreign | 5 | 3 | 60.0 |
| Total | 202 | 35 | 17.3 |

It is evident, then, that Freetown itself enjoys a comparative immunity from infection, but what the probable explanation of this is I am not yet prepared to say. There is a large aboriginal population in the city, and a constant stream of natives coming from and going to the Protectorate, a large proportion of whom must be infected, and it would seem likely that the townspeople would get infected from this source. Possibly one reason may be the distribution of the people in the town, the aboriginals living, as a rule, in one quarter, while the natives of Freetown live in another, but this is by no means a hard-and-fast rule; or perhaps the variety and distribution of the mosquitoes in the city have a good deal to do with it. This point I have not yet been able to work out. The fact is a very interesting one, that we have a community composed almost entirely of negroes, one class of which, so far as my figures go, are comparatively free from filarial infection, while the others suffer to such a large extent.

The following table shows the varieties of filaria met with:—

TABLE III.

| | No. Examined | Filaria Nocturna | Filaria Diurna | Filaria Perstans | Filaria ? | Filaria Nocturna & Perstans | Filaria Nocturna & Gigas |
|-----------------|--------------|------------------|----------------|------------------|-----------|-----------------------------|--------------------------|
| Bandajuma | 20 | 6 | — | — | — | — | — |
| Moyamba | 31 | 11 | — | — | — | — | 1 |
| Sherbro | 22 | 5 | — | — | — | — | — |
| Freetown | 202 | 28 | 2 | 2 | 2 | 1 | — |
| Total | 275 | 49 | 2 | 2 | 2 | 1 | 1 |

(1) It will be seen that, as is generally the case elsewhere, filaria nocturna predominates. In the majority of instances where filaria were met with both day and night, those in the daytime were few in number, and were evidently stray nocturna. In one case there was a double infection of nocturna and perstans, and in another nocturna and a new filaria.

(2) Filaria diurna was found in two cases, with physical characteristics much the same as nocturna. I am inclined to believe, however, from measurements of stained specimens (though I am aware this is apt to vary), that filaria diurna is a slightly smaller worm than nocturna, and that the tail does not taper so sharply.

(3) Filaria perstans occurred in three cases.

(4) In two cases I found a minute filaria measuring 0.112 millimetres in length by 2 to 3 μ in breadth, or less than half the size of filaria nocturna. It has a rounded head and a truncated tail. There is a clear linear band about one-fourth of the length from the head, a clear spot a little further down, another irregular clear spot about two-thirds of the distance, and a linear mark near the tail. I am unable to identify this worm, but it appears to me to resemble very nearly the worm which has been described by Dr. Manson as filaria Ozzardi. It has been suggested to me that it is a perstans very much shrunk. Although I know that perstans is apt to vary in size, yet the difference in size seems to be more than can be accounted for in this way, and in those cases where it was found all the filaria were the same size. I am inclined to regard it as a different species.

(5) In one specimen I came across a worm which, so far as I know, has not hitherto been described. The most striking characteristic is its great length and thickness, 0.34 mm. by 8 to 12 μ . It stains very readily with fuchsin, and retains the stain with great tenacity, so much so that I was unable to decolourise it with glacial acetic acid without spoiling the specimen. The structure is consequently obscured. There were two specimens in the blood film (which also contained nocturna), one much larger than the other, the small measuring 22 mm. by 8 to 10 μ . The head is rounded, there is a taper to the tail, which is blunt, and no sheath could be made out. On careful focussing, evidence of a central canal could be made out, but beyond this description I am unable to go at present. Of course I am not in a position to say what the pathological significance of such a large worm in the blood is. It occurred in a private of the Frontier Force, apparently in good health, so far as I have been able to ascertain, and I hope to obtain further specimens of his blood. In the meantime I place it on

record, and suggest, provisionally, the name of "*filaria gigas*."

(6) *Filaria volvulus*.—I have already described the adult male, female, and embryos of this worm.¹ They measure 25 mm. in length by 5 μ in breadth, have a marked clear spot, a central granular aggregation, a pointed tail, and no sheath.

(7) *Filaria loa*.—This was a very interesting case. The patient, a European under the care of Dr. Renner, to whose kindness I am indebted for the opportunity of seeing the case, had had two adult worms removed, one from the loose skin of the penis and the other from the eyelid. He had been resident at Sherbro for about a year, but some time previously had been living on the Congo. On examining the blood it was found to be swarming with an embryo which is presumably that of *filaria loa*. It was about 3 mm. in length and 3 to 4 μ in breadth. It has a taper to the tail, which is pointed, but I should say not so sharply as that of *filaria nocturna*. There are four clear spots, but the swellings along the course of the worm I am inclined to attribute to changes during preparation. I have not myself seen a case of *filaria loa* previous to this in Sierra Leone, and it is probable that it was acquired while in the Congo.

Filarial diseases.—The prevalence of diseases which are actually filarial in origin is difficult to arrive at. The following table shows the different diseases from which patients in the Colonial Hospital, in whose blood *filaria* were found, were suffering :—

TABLE IV.

| Disease | Nocturna | Diurna | Perstans | Filaria (?) | Nocturna and Perstans | Nocturna and Gigas |
|------------------------|----------|--------|----------|-------------|-----------------------|--------------------|
| Injuries | 9 | — | — | — | — | — |
| Ulcer | 6 | — | — | — | — | — |
| Necrosis | 1 | — | — | — | — | — |
| Bronchitis | 1 | — | — | — | — | — |
| Diarrhoea | 1 | — | 1 | — | — | — |
| Melancholia | 1 | — | — | — | — | — |
| Conjunctivitis | — | — | — | — | — | — |
| Fibroma | 1 | 1 | 1 | — | — | — |
| Debility | 4 | — | — | — | — | — |
| Syphilis | 1 | — | — | — | — | — |
| Rheumatism | 3 | — | — | — | 1 | — |
| Fracture | 1 | — | — | — | — | — |
| Peripheral neuritis .. | 1 | — | — | — | — | — |
| Peritonitis | — | 1 | — | 1 | — | — |
| Stricture | 1 | — | — | — | — | — |
| Lipomata | — | — | — | 1 | — | — |
| Diseases not recorded | 8 | — | — | — | — | — |

It will be seen that the most common diseases were ulcer, debility and rheumatism. I consider it not improbable that the vague aches and pains which are classed as rheumatism, and which are so common in Sierra Leone, may be associated with the presence of *filaria* in the blood, and that the ulcers which reach such a large size in natives may have some relation to obstruction of the lymphatics.

Chyluria has been noted, but is rare. Lymph scrotum is also rare. Enlarged lymph glands, especially in the groin, are very common. Elephantiasis cannot be said to be a common disease. In 1,210 cases admitted to

the Colonial Hospital in 1900 there were no cases of elephantiasis; in 1901, out of 1,265 there were two cases of elephantiasis of scrotum and two of the leg. In the Protectorate, where, as I have shown, *filaria nocturna* is very prevalent, elephantiasis does not appear to be common. In the absence of exact information as to the population, it is difficult to get a definite idea as to the percentage, but one of the district medical officers, who has now had a large experience, considers that 5 per 10,000 is an outside estimate, while others agree that it is by no means common.

The question of the relationship of elephantiasis to the presence of *filaria nocturna* in the blood appears to me to rest on a very slender and almost entirely theoretical basis. One of the principal arguments is that the distribution of *nocturna* and elephantiasis coincide, and that the one increases in the same ratio as the other. I venture to think that the present instance is one which militates strongly against this argument, for if the presence of *filaria nocturna* is the cause of elephantiasis, we should naturally expect that where the percentage of infection is so high the prevalence of elephantiasis would be greater than it is; and I am inclined to believe that as the geographical distribution of *nocturna* is more worked out it will be found that it is more extensive than is supposed.

Space forbids me to deal with the other arguments which are adduced in favour of this theory; but though I do not go so far as to take up the position that elephantiasis may not be due to *nocturna*, it seems to me that the almost universal acceptance of this pre-conceived and unproved theory has had the unfortunate result of putting a stop to investigations along other lines.

THE CAUSE OF BERI-BERI.

By CAPTAIN E. R. ROST, I.M.S.

Having worked at the pathology of this disease since 1898, and having conducted a large number of experiments, already published in the *Indian Medical Gazette* for December, 1900, July, 1901, and July, 1902, I am anxious to bring the matter up for discussion, hoping that someone may verify the experiments.

An outbreak of beri-beri occurred at Meiktila Jail in 1898, during which I observed that pigeons (which lived in large numbers under the roofs of the Jail buildings) were affected by an epidemic disease, which caused paralysis of the wings and death. At this time I thought I had traced the disease to a micrococcus which I found in the jewari, the staple food of the Jail. Between the tiles of the roofs and the boards, accumulations of pigeons' excreta of years' duration was found and cleared out, wire-netting being put up to prevent the pigeons again nesting. After the pigeons had been thus circumvented the epidemic died out. The jewari was stocked yearly in the granary and the lower layers were found mouldy and were condemned.

Later, in coming to Rangoon, I had the opportunity of studying this disease and was brought by my former experiences and a study of the etiology of the disease in Rangoon, to search for the causation in rice. I found in rice-water liquor and in mouldy rice an

¹ *British Medical Journal*, January 26th, 1901, p. 209.

angular diplo-bacillus between the starch granules, and found that this was extremely resistant to high temperatures, a temperature of 220° F. for nine hours being necessary to kill off the spores of the diplo-bacillus. I found this organism in the blood and cerebro-spinal fluid of a large number of beri-beri cases; the blood was removed by the pipette method and cultivated in broth, rice-broth and ascitic fluid. It was a sporulating angular diplo-bacillus which would thus appear in all varieties of shapes, it was very mobile, easily stained by carbo-fuchsin, and about the size of the tubercle bacillus. I then commenced injection experiments from cultures of this organism into fowls. They died with symptoms to be hereafter described and the bacillus was found in their blood and spinal cords.

I have now carried out a very large number of experiments on fowls and latterly on pigeons, and will mention these in detail, as they are most convincing in showing the connection between a disease in rice and beri-beri in man. An exactly similar disease was produced in fowls:—

- (i.) By feeding them on fermenting rice obtained from the rice-liquor shops.
- (ii.) By feeding them on mouldy rice obtained from the lower bags of damp godowns.
- (iii.) By intraperitoneal injection of rice-water liquor.
- (iv.) By subcutaneous or intraperitoneal injection of the venous blood of beri-beri patients.
- (v.) By reinjection from fowls suffering from the disease produced as above.

(1) Fowls fed on fermenting rice obtained from pegu jars in the rice-liquor shops of the town develop a disease which is rapidly fatal. Out of thirty fowls experimented upon in this direction not one recovered, although three removed from the influence of the feeding died shortly after. The symptoms came on gradually with loss of feathers, anæmia, diarrhœa, weakness, listlessness, and great loss of weight. The feathers commenced to fall out on the neck and extended to the trunk, the cockscombs became blue. There was some suspicion of ataxia, as some would fall over on being chased. They fed well of the rice until the final stage of paralysis occurred. The time at which the fatal termination would come on was very variable, and appeared to depend on the weight of the bird. At the paralytic stage they stood in crouched-up positions supporting themselves on their backs and tails, with their feet in the air, their wings spread out to balance themselves. On being thrown up they fell like dead weights. They became paralytic and ataxic, and later cyanosed, and gasped for breath. Some died very rapidly, others took about two days to die in this condition.

In some the diarrhœa was severe, and in some cases there were bloody, slimy evacuations. The *post-mortem* appearances showed hyperæmia and thickening of the gastro-intestinal tract, in some cases marked petechiæ in the small intestines (Professor A. Holst in a large number of autopsies performed by him in Rangoon on beri-beri cases found thickening and petechiæ in the small intestine, though this was also found in cases of diarrhœa and dysentery).

Latterly, three fowls were injected subcutaneously from the heart-blood of three fowls dead of the disease

by feeding on fermenting rice; these birds all died with the same symptoms: there was less loss of feathers, but the same great anæmia, loss of weight, and finally the paralytic stage. To contrast this condition with similar remove experiments carried out from fowls dead of the disease after the injection from beri-beri blood (suggested to me by Professor Holst), made it quite impossible to doubt that the two conditions were but one and the same disease.

(2) In very much the same way, fowls fed on mouldy rice, from which the fermenting rice above referred to is prepared, suffer from the symptoms above described, the diarrhœa is less severe and course of the disease more prolonged, but the fatal termination the same.

(3) Three fowls were injected intraperitoneally with rice-water liquor, and died about two months later, after diarrhœa, loss of feathers, great anæmia, the fatal termination being the same as in the other cases.

(4) Twenty-four fowls were injected intraperitoneally from the venous blood of beri-beri patients; all died with the same symptoms except two, in which the disease did not develop.

The blood was removed from the median cephalic vein by insertion of a sterile glass pipette directly into the vein, sealing the ends, breaking off the ends, and inserting the pipette into the abdomen of the fowl direct, the blood from the pipette running into the abdomen cavity.

These birds showed symptoms which came on gradually: diarrhœa, anæmia, loss of feathers, weakness, drooping of the wings. They fell rapidly in weight, but partook of their food well.

The acute symptoms would suddenly come on and the bird die with symptoms identical as in the fermenting rice series.

Post mortem showed hyperæmia and petechiæ in the small intestine (this was only looked for in the later repetition of the experiments).

Several control fowls injected with blood of other diseases did not develop the disease. Professor Axel Holst was at this time experimenting on the subject of beri-beri in Rangoon, and at his instigation we tried the effect of injecting a small amount of blood from a fowl dead of the disease into three healthy fowls. These were injected subcutaneously with a broth emulsion of a small amount of the blood of a fowl dead of the disease. These three fowls died of the disease, and from the blood of these other fowls were injected subcutaneously. The same symptoms appeared in these, a third and a fourth series being injected, all dying with the symptoms already described.

Latterly the same experiments with pigeons were tried. Six pigeons injected intraperitoneally with the blood of beri-beri patients developed symptoms which came on suddenly from one week to three weeks after the injection. They fell in weight, had diarrhœa, anæmia, could not fly or stand, became cyanosed and gasped for breath, and died. *Post mortem*: hyperæmia and petechiæ were found in the small intestines. Reinjection has produced the same symptoms in another pigeon now under observation.

The later experiments of reinjection show that the disease must be caused by a micro-organism in the blood; that fowls fed on diseased rice die from a

disease due to a micro-organism in the blood, which, on reinjection, produces the same disease. That the birds injected from the blood of beri-beri patients die from a disease due to a micro-organism in the blood, which, on reinjection, produces the same disease. From the identity of the symptoms of the disease produced in these two ways, and the identity of the symptoms of reinjection in both instances, one can have little doubt that the two conditions are caused by the same micro-organism.

I will now draw your attention to the etiology of the disease in Rangoon, which in itself is so remarkably in favour of the rice origin, as to leave little doubt as to the causation of this disease.

An analysis of 390 cases treated as in-patients in the General Hospital, Rangoon, in 1901, show that 342 were Hindus, 364 were males, 338 were coolies. The usual age was between twenty and forty years. No case in a child has been known, and cases in women are comparatively rare. Captain Barry, I.M.S., in the *Indian Medical Gazette* for September, 1900, has gone into this matter thoroughly, and shows how the cases increase with immigration of the Hindu coolie.

The Hindu coolie is the man who is chiefly addicted to rice-liquor drinking; he is very fond of it, and can obtain a quart for one pie. They are not the only people, however, who drink it, Madras Sepoys, even British troops, Eurasians, and sometimes women.

In Lower Burma there are special shops where this liquor is exclusively prepared and sold. I have been to several of these shops, seen the liquor made and the class of man drinking. It is prepared from bad rice bought at a cheap rate from paddy brokers, who in bringing paddy to the mills in the Poozondanug Creek, get their boat loads occasionally damaged by water; the mill-owners refuse this, and the paddy broker sells off at a cheap rate. This bad rice is made worse by storing in damp godowns.

The rice-liquor is made by allowing this damp rice to ferment in pegu-jars, which are never cleaned out, the water merely being poured off into bottles, which are sold across the counter. The longer the stuff is allowed to remain in the bottles the better it is appreciated. It tastes like cider.

The chemical analyst's report on this liquor shows that it contains phosphoric acid and sodium chloride in small quantities, acetic acid, and alcohol (7 per cent. absolute alcohol by weight).

No authentic case is on record in this hospital in which a man is said to have contracted the disease in hospital, some have contracted the disease in hospital, but these have been in the habit of going out to the bazaar or have not been long in hospital. The coolie classes in Rangoon live together in an extremely crowded state, yet no cases have been found in children and rarely in women. No particular area or group of houses has been known to be affected, and it has never appeared as an epidemic here.

With regard to the symptoms, in going through the cases of 1901, I find that out of 390, 202 had a rise of temperature or diarrhoea, 90 had both a rise of temperature and diarrhoea. In 119 cases the diarrhoea was severe and lasted throughout the illness. The rise of temperature was irregular and not thought to

be of malarial origin. Moreover, blood examinations in the cases did not reveal the plasmodium.

I mention these two symptoms, recalling the *post-mortem* appearance of the intestine of beri-beri cases and of beri-beri fowls, as showing that the primary focus of infection is in the intestine. There are some instances of outbreaks of this disease in lightships, in schools, and in countries where it would appear difficult to look for the causation of the disease in rice-liquor or in diseased rice. But it is probable that rice is not the only cereal in which this disease can grow; moreover, it is possible that the disease may be, in some instances, communicated by means of fowls suffering from the disease. This reminds me that I have seen fowls suffer from a disease accompanied by loss of feathers and diarrhoea and I am told that fowls do suffer from epidemics of diarrhoea and loss of feathers, that the disease lasts some weeks, and is, therefore, unlike fowl cholera.

THE PROPHYLAXIS OF SUNSTROKE.

By ANDREW DUNCAN, M.D., B.S., F.R.C.S., M.R.C.P.

Physician, Seamen's Hospital Society and Westminster General Infirmary.

HAVING unfortunately during my career in India suffered on four occasions from the effects of the sun, I venture to detail a method of preventive treatment which answered admirably in my case. I will first sketch briefly the clinical symptoms of my attacks. In August, 1880, I had returned to India in the rudest health after six months' sick leave to New Zealand from Cabul. I was sent up on service again immediately, but this time on the Candahar line. I reached Sibi in the first week of September. What the temperature must be at this place in July is rightly estimated by the saying of the natives of this part of the country, when they wonder why the Supreme Being made hell when Sibi was at hand on the earth. In September, 1880, the train came abruptly to a stand on a hot, dusty plain, no railway station being as yet built. In walking over to the dak bungalow, even at this time of the year, the heat was terrific. The same week I joined my regiment in the Pishin Valley, and the next day we took part in a small expedition against some recalcitrant village. On returning to camp my head was splitting, and I had to be sent at once to the hospital. During the next twenty-four hours the pain continued to increase, accompanied by great intolerance to light, and I was fast lapsing into unconsciousness, when, fortunately for me, my friend, Surgeon Armstrong, saw me, had my head shaved and blistered, and practically saved my life. For the next six weeks, however, the racking pain in the head never left me, except at the end of this time, in the evening hours. I then was invalided to England, but it was not until I got west of Suez that the headaches ceased. After two years' sick leave I returned to India, and was stationed at Mian Mir. About March, as the hot weather commenced, I was again seized with these distressing headaches, and in the first week of this second onset a temporary attack of hemiplegia

occurred. I was invalided to Kashmir for five months. During the first four months of my sick leave I scarcely remained for a day free from intense pain in the head. After four months the pains gradually ceased, and I returned to duty. Each succeeding hot weather for a series of years severe headaches now regularly tormented me, but no definite attack of sunstroke occurred until 1887, when I was again the victim of this affection whilst on leave shooting in the Himalayas. Lastly, in April, 1891, whilst on the Black Mountain expedition, my last attack occurred, characterised by the same distressing symptoms—namely, an intolerable headache, &c. For this I had five months' sick leave to the hills, but, on returning to duty in the plains I was again knocked over by the sun, and had to take two years' leave to England. Shortly after my return to duty, I fortunately read a letter in the *Pioneer* newspaper, written by an executive engineer. This officer had suffered on several occasions from sunstroke. Reasoning from the fact that no one gets heat-stroke from the great heat of furnaces in an arsenal, he came to the conclusion that the heat rays of the sun were not the *fons et origo mali*, but the actinic rays. Hence he treated his body like a photographer treats his plates, and enveloped it in orange, using always an orange-yellow shirt, and lining his coat and hat with flannel of the same colour. During the subsequent five years of extreme exposure he suffered from no bad effects of the sun. Acting on this hint, whenever I had to perform a march in the hot weather I always used an orange-yellow shirt; I lined my helmet with orange-yellow flannel, and had a pad of the same colour stitched into my khaki coat down the back. I, too, never again, in consequence, felt the effects of the sun. I would, therefore, submit that the dangerous rays of the sun are the actinic, and as a means of protection from sunstroke I would advocate the use of such means as I have indicated. As a further precaution the helmet might be lined with a layer of tinfoil.

News and Notes.

THE QUARANTINE STATION OF EL TOR.

By one of the Nursing Staff.

THE pilgrimage to Mecca has been a very large one this year, about 100,000 pilgrims visiting the sacred shrine, of these nearly 40,000 coming from the north. Now that the season is practically over, an account of the measures that have been successfully taken to fight the threatened epidemic of cholera may prove of interest. Two objects have been kept in view; one to prevent any pilgrim from returning to Egypt without passing through Tor, and the other to stamp out the epidemic by fighting it in the most efficacious manner at the quarantine station, and allowing the pilgrims to leave only when they are considered incapable of carrying infection. On the arrival of steamers the pilgrims are visited by the quarantine doctors, all

healthy pilgrims are disembarked in large boats, towed to the landing stages and taken to the sections. A section forms a rectangle, 200 metres long by 50 metres wide, enclosed by a high fence and separated on both sides from the next section by a vacant plot of similar dimensions. The ground is laid out in four rows of 25 tents, each tent to hold six pilgrims. At the corners are the tents for the doctor, sanitary guards, and the representative of the Ministry of the Interior. Each section contains a general provision store, a restaurant, and the necessary sanitary provisions. The pilgrims are visited individually twice a day by the doctor of the section, who sends any sick to hospital. In cholera times the period of quarantine is fifteen days, provided that the health of a section remains good; but if at any time a case of cholera occurs in a section the whole period of quarantine begins again. The sick are, however, conveyed straight to hospital and the dead brought ashore and buried. Then the ships and everything on board are thoroughly disinfected and during the rest of their stay are constantly visited by the doctors.

Disinfecting the Pilgrims.—The camp consists of bathing and disinfecting houses, sections, hospitals, offices, store rooms, dairy, tents for the president, the staff, the servants, and the soldiers. When the healthy pilgrims from a ship are disembarked they are taken first to the bathing and disinfecting houses, one of which is reserved for women, and is in charge of an Englishwoman. Their goods and clothing are put in numbered sacks, which are subjected to a heat of 120° F., for twenty minutes in the disinfecting ovens; but everything which would be spoilt by heat is soaked instead in a solution of corrosive sublimate. Meanwhile the pilgrims are taken into spacious dressing rooms and there provided with long calico garments before passing on to the bath-rooms. Here they have a choice of hot or cold douche, or hot or cold sea-water bath, for which a special soap is provided. The bath over, they go on to adjoining rooms, where their disinfected clothes and goods are handed back to them, and while they are dressing the quarantine doctor visits them, and a list of their names is taken by the passport authorities. The pilgrims are passed through in batches of forty, each batch occupying about an hour. After these preliminaries are gone through, the goods are put upon a small train kept running between the different parts of the encampment, and all the pilgrims from one ship follow to the section destined for them.

The Hospitals.—There are three hospitals built of masonry, two containing 30 beds each and a small bedroom for the doctor in charge, and the third containing two wards of 10 beds, one ward reserved for women and one for surgical cases. There is a well-appointed, up-to-date little theatre, and also two extra rooms which came in very useful this year for members of the staff who, unfortunately, broke down. Near the gate of the hospital enclosure is the bathing and disinfecting house, and close by is the laundry. There is also a well-fitted bacteriological laboratory. There is an enclosure fenced off and containing wooden huts with adjustable ventilating shutters, and containing two beds each. This was used for isolating cholera and other contagious cases. There is yet another en-

closure containing tents for housing suspected cases. As all these different enclosures became full it was necessary to erect tents, until there were about 50 with two patients in each. The staff consists of the director, five or six other doctors, and a bacteriologist, trained European sisters (English, German, and Greek), and Arab nurses, both male and female.—*Extract from 'The Hospital,'* August 2nd, 1902.

Current Literature.

CHINESE versus EUROPEAN RHUBARB.—Dr. S. Jakabházy describes the differences in markings which may serve as points of distinction between the two kinds. European rhubarb is characterised by relative freedom from crystalline glands and comparative richness in starch. There is a difference, too, in mineral matter which in Chinese rhubarb amounts to from 8 to 25 per cent., being only 1·3 to 6 per cent. in European species. The safest means of discrimination is, however, a quantitative determination of the essential principles. The author found as follows:—

| | | Extract by Ammoniacal Alcohol | Chrysophanic Acid | Emodin | Pseudo-emodin and Pseudo-frangulin | Frangulic Acid | Double Glucosides |
|----------|-------|-------------------------------------|----------------------|--------|--|----------------|----------------------|
| Chinese | I. | 47·3 | 3·71 | 1·70 | 2·64 | 3·91 | 21·2 |
| " | II. | 39·5 | 2·92 | 1·31 | 2·33 | 3·21 | 22·3 |
| " | III. | 41·2 | 3·07 | 1·43 | 2·19 | 2·87 | 19·6 |
| English | IV. | 36·3 | 1·86 | 0·59 | 1·36 | 1·88 | 20·5 |
| " | V. | 33·5 | 0·80 | 0·38 | 1·21 | 1·04 | 15·3 |
| Austrian | VI. | 27·5 | 0·54 | 0·41 | 0·69 | 1·70 | 14·7 |
| " | VII. | 30·7 | 0·70 | 0·47 | 0·83 | 2·02 | 19·3 |
| French | VIII. | 31·2 | 0·74 | 0·38 | 0·68 | 1·71 | 16·4 |

The Chinese are seen to be far richer in chrysophanic acid and emodin than are the European.

In reference to the above "Double Glucoside," a paper of importance—bearing in view the wide use of the drugs in question—appeared in the *Apoth. Ztg.* (May 31st, 1902), by Dr. Aweng, describing his method of isolation and the properties of the compound. Briefly, he prepares firstly an aqueous extract, which after concentration is treated with four volumes of absolute alcohol and the filtered solution evaporated to small bulk, the residue dissolved in cold water leaving rhamnetin and pseudofrangulin in the insoluble portion. The aqueous solution again concentrated is taken up with a large volume of absolute alcohol, the precipitate formed containing frangulic acid (a non-glucosidal body yielding by acid treatment two rhamnetin-like substances). The alcoholic solution contains the double glucoside of which the yield is 5 to 6 per cent. This compound in the first stage of hydrolysis splits into a sugar, a little emodin and chrysophanic acid, and further a substance corresponding to the author's pseudo-emodin (Tschirch's *Nigrin*, Leprince's *Cascarin*); this in turn is a compound of emodin chrysophanic and frangulic acids.

More severely treated (two hours' boiling with HCl), the double glucoside is decomposed into much emodin, some chrysophanic acid, and the two frangulic acid decomposition products, whence it would appear to have a very complex composition. It affords a safe, sure and mild purgative, and for galenical use the author recommends an extract made with 80 or 90 per cent. alcohol. The glucoside is very sensitive to both acids and alkalis.

A somewhat similar compound has been separated not only from frangula bark and rhubarb, but also from senna and sagrada.—*Zeits. d. allg. oesterr. Ap. V.*; also *Apoth. Ztg.*, 1902, 358.

A CLINICAL, BACTERIOLOGICAL AND ANATOMO-PATHOLOGICAL CONTRIBUTION TO THE STUDY OF LEPROSY OF THE NERVOUS SYSTEM, OF SYRINGOMYELIA AND OF MORVAN'S DISEASE.—Dr. Calderone sets forth his experience respecting leprosy and Morvan's disease. He comes to the conclusion that Morvan's disease is a form of leprosy, and supports this assertion by his discovery of Hansen's bacillus in the lesions of the disease.

The differentiation of syringomyelia from leprosy is not easy in countries where leprosy prevails. Notwithstanding the fact that sensory dissociation is found in both diseases, the author considers syringomyelia is a specific ailment. He advises particular attention to maculæ of the skin, enlargement of the spleen, and intermittent fever as the differential diagnosis of leprosy.—*Giorn. Ital. delle Mal. Ven. e della Pelle*, 1901, fasc. vi., p. 756.

CONGENITAL MALARIAL FEVER.—Dr. Lindsay Peters reports the case of a child born at term during a malarial paroxysm of the mother. After reviewing the literature, the author says that since it is well proved that many varieties of bacteria are able to pass from the maternal to the foetal circulation, it is *prima facie* likely that "a minute organism possessing the active amoeboid motility and penetrating power of the malarial parasite should be able to do so." The mother had been once cured of tertian fever. The attacks recommenced in the spring of 1898, she being at the time pregnant, but again disappeared under treatment, to return on the day of confinement. On the third day the mother had a chill and a temperature of 104° F. An examination of fresh blood specimens the next day showed typical, half-grown, tertian malarial organisms. The baby was a healthy boy. Blood from the ear showed no organisms on the first two days. On the fiftieth day after birth the child was seen and found to be sick, pale, unwilling to suck, anæmic, and yellowish. The lower border of the spleen could be felt. The next day a specimen of blood was pale, watery, and coagulated very slowly. Three malarial parasites of the tertian type were found—one small, extracellular, pigmented form, and two half-grown intracellular forms containing light brown dancing pigment. Quinine removed the anæmia and other symptoms in a few weeks. The child was reported to have "got cold and had fevers" from the second week after birth. In infants well-marked chill is said to be rare, and to be replaced by "cold hands and feet, blue lips and nails, sometimes slight

general cyanosis, pallor, drowsiness, and prostration." From these considerations the author concludes that the affection was not recent, but had been acquired *in utero*, or very soon after birth. The latter was considered highly improbable, and the author inclines to the view of intrauterine infection.—*Johns Hopkins Hospital Bulletin*, June, 1902.

A new reaction for quinine and quinidine has been discovered by E. Hirschsohn, and consists in the addition of one drop of hydrogen peroxide solution (about 2 per cent.) and one drop of 10 per cent. cupric sulphate to the neutral alkaloidal solution (chloride or sulphate); on boiling, an intense raspberry-red coloration appears, changing to bluish-violet, then blue, and after a while, slowly to green. The limit of sensibility is 1 in 10,000. As in every such test it is highly important to know upon what other substances it has been tried, the following list increases the value of the reaction very considerably. A colourless or only faintly yellow solution is obtained with antipyrine, atropine, aconitine, acolyctine, asaron, brucine, berberine, bebeerine, caffeine, cocaine, cinchonidine, cinchonine, cinchonamine, cinchotenine, codeine, colchicine, colocynthin, convallamarin, convallarin, coumarin, cubebin, caryophyllin, delphinin, daturine, digitin, digitalin, duboisine, gelsemine, helenin, hyoscine, hyoscyamine, kosiin, meconin, minispermene, piperine, picrotoxin, pilocarpine, quassiin, quinoline, solanine, saponin, santonin, salicin, senegin, scoparine, sabadilline, sparteine, strychnine, taxine, theobromine, urson, vanillin, veratrine. The following give yellow to brown: Analgene, apomorphine, arbutin, æsculin, chelerythrin, cotoin, duboisine (amorphous), eserine, hydrastine, hydrastinine, morphine, narceine, narcotine, paracotoin, papaverine, peucedanin, phloridzin and pyrodine. A light onion-red is produced by thalline, an intense blue by kairine, and a light blue by asparagine. The peculiar raspberry-red tint is observed with equinine, but only feebly, so that this colour when strongly developed seems quite characteristic of quinine or quinidine. — *Pharm. Centralt. Sud. d. Ap. Ztg.*

CHOLERA IN EGYPT.

During the week terminating September 22nd, 6,388 cases of the disease were reported in Egypt, and 4,022 cases during the week ending September 29th, thus showing a decrease of 2,366 during the latter week: 1,932 towns, villages and ezbehs are now reported to be infected in Egypt, showing an increase of 375 during the last fortnight.

PLAGUE.

PREVALENCE OF THE DISEASE.

India.—During the two weeks ending September 20th, the total number of deaths from plague was 15,569. The districts most severely affected include the Bombay Presidency, Mysore, the N.W. Provinces, and Oudh. The epidemic is spreading rapidly, and Bangalore City and Kolar Gold Fields are now involved. In the Punjab the mortality for the week ending September 13th was 101, and for the week ending September 20th, 90—a decrease of 11.

Egypt.—During the week ending September 21st there was only one case reported and no death, and during the week ending September 28th there were four fresh cases and two deaths. All the cases occurred in Alexandria.

Mauritius.—During the week ending October 2nd there were nine cases and eight deaths, and during the week ending October 9th, fourteen fresh cases and eleven deaths.

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Archives Russes de Pathologie, de Médec. Clinique et de Bacteriologie.
Australasian Medical Gazette.
Boletín de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Brooklyn Medical Journal.
Caducée.
Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Esercito.
Hong Kong Telegraph.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
Journal of the American Medical Association.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Medical Record.
Medical Review.
Merck's Archives.
New York Medical Journal.
New York Post-Graduate.
Pacific Medical Journal.
Polyclinic.
Public Health.
Revista de Medicina Tropical.
Revista Médica de S. Paulo.
Sei-i-Kwai Medical Journal.
The Hospital.
The Northumberland and Durham Medical Journal.
Treatment.

Notices to Correspondents.

- 1.—Manuscripts sent in cannot be returned.
- 2.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.
- 3.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.
- 4.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.
- 5.—Correspondents should look for replies under the heading "Answers to Correspondents."

The Journal of Tropical Medicine.

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Original Communications.

NOTES OF A TOUR IN THE NORTH CANARA DISTRICT OF INDIA IN SEARCH OF MOSQUITOES.

By E. H. AITKEN.

LAST March I was deputed by Government to visit some of the Customs posts on the frontiers of the Portuguese territory of Goa, and to see whether anything could be done to reduce the malaria from which our guards suffer. The frontier line begins near to the old seaport of Vingorla in the Rutnagherry district, and strikes inland till it reaches the top of the Ghauts, when it turns south and runs for a distance of about fifty miles, as the crow flies, along the crest of the ascent, at a distance from the sea ranging between twenty and thirty miles. Then it turns west and descends to the level of the sea, which it reaches a little south of Karwar in the North Canara district. The country through which it passes, especially towards the south, is to a great extent primitive forest, the home of the tiger, the black bear and the bison, and of many striking birds, such as the Great Hornbill and the Racket-tailed Drongo. It is inhabited by a race known as *Coonbie*. What connection there is between them and the Coonbies who constitute the ordinary peasant class of the Deccan is an undetermined question, but these more resemble the aboriginal hill tribes in many respects. They practise what is called *Coomrie* cultivation, as far as they are allowed, cutting down and burning much forest for a little coarse grain, and they also cultivate rice wherever they can find a few acres of level ground contiguous to a stream. Off this they raise two crops, one in the monsoon and another in the hot season. This hot-season rice is responsible, as will be seen further on, for much of the malaria with which this beautiful region is afflicted. It is a fact which those acquainted with other parts of India have difficulty in believing, that the fever season here is not September and

October, but April and May. The reason is not difficult to divine. The rainfall is exceedingly heavy, amounting to 300 or 400 inches a year, and during the monsoon and for some months after it all the rivulets and streams are rushing at a rate which makes larval life impossible. From November to February, moreover, the cold at night is often severe. But by the month of March the weather has begun to get warmer, and the streams have shrunk and got broken up into detached pools, the process being aided by the masses of dead leaves which have fallen from the forest. Thunder-storms are also frequent at this period, filling every small depression in the ground where they occur, but too local to flood the streams. And by this time also the natives have diverted the water of many perennial streams into their fields, and by a skilful arrangement of dams, converted these into still, but not stagnant, pools a few inches deep. In these hot season rice-fields there appear to be few, if any, fish, and there are plenty of weeds to afford larvæ cover from their enemies. It is no wonder, therefore, if this is a season of prosperity and increase for the whole mosquito tribe, and also for the parasites of malaria.

Having been in this district before, I was partly aware of these conditions, and so chose the month of March for my tour, but I was delayed, and so did not reach Castle Rock till the 27th of that month. Castle Rock is the frontier station of the Portuguese Railway, where it connects with the Southern Mahratta line, and I decided to begin here and go southwards, the southern half of the frontier being much more malarious than the northern. Castle Rock is not considered a malarious station, the houses being mostly built on high ground and well exposed to the wind. I caught several specimens, however, of *A. christophersi* (Theobald) on the second day, in a cowhouse, and also in a shed in which goats were kept. I may mention here that throughout this trip I had very little success in catching mosquitoes. If this had been true only of the wily *Anopheles* I might have

ascribed it to my own want of skill or patience; but it was equally true of *Culex*. In camps at which the servants complained that they had not been able to sleep owing to the persecution of "mutchers,"¹ I failed in the morning to find a single winged thing. My inference is that in open country mosquitoes are compelled to spend the day in houses for shelter from the sun, but that when cool and shady jungle is near at hand they prefer to retire into it. That there were mosquitoes in plenty was proved by the number of larvæ which I found. Within two days I had collected a most puzzling variety of forms, many of which were quite strange to me. I was unprepared for this and unprovided. Knowing the difficulty of transport in these wilds, I had reduced my luggage to absolute necessities, and the few soup plates, saucers and finger-bowls I could muster hardly sufficed to accommodate those varieties of larvæ which it was most important to keep separate. And in the middle of some critical observation my boy would come and demand the saucer because it was tea-time. When I travelled it was still more difficult to find bottles to hold all my menagerie. All travelling had to be done on foot and all baggage carried on coolies' heads. As my time was limited, I had often to march on consecutive days, in fact, I moved six times in the course of one week. The result of all these embarrassments was that I lost some valuable specimens, and failed to rear some interesting larvæ. If I ever undertake such a tour again, I will provide myself with a dozen white saucers and as many wide-mouthed bottles with good stoppers.

To return to Castle Rock. I spent five days there collecting, rearing and trying to discriminate. I also visited Anmod, a small outpost, five miles distant, at which our men were said to be suffering much from fever at the time. I found that their lines were situated within a hundred yards of extensive rice-fields, in which, after much fishing with a white teacup, I found numerous larvæ of *Culex fatigans* and of an *Anopheles* which I did not recognise at the time and lost afterwards. They were probably *A. christophersi*, which I found to be the commonest larva in rice-fields elsewhere. I could devise no way of "curing" a rice-field, so I recommended the removal of the lines to an airy site at some distance, and I believe this is to be done shortly.

The next post with a bad reputation was Diggi, situated on one of the principal trade routes from Goa, and consequently an important station with a considerable staff. At Castle Rock I had enjoyed the hospitality of Mr. Pogson, the Assistant Collector in charge of the whole frontier, and, as he had business at Diggi also, we travelled together. I need scarcely say that the society of another white man was welcome in these ever delightful, but weirdly solitary, wilds. At Diggi we found that both clerks and peons had been suffering from fever for some time past, and it did not take long to find the sources of infection. Not far from the lines behind there was a small stream flowing through dark jungle. At one point, where it crossed a path, it had been dammed so as to form a pool at which the cartmen might water their

cattle, and here I found larvæ at once. Most parts of the stream were too shady for them. In front of the lines and somewhat further away there was another stream flowing into rice-fields, and in the middle of these a small tank, absolutely choked with watergrass and swarming with larvæ, from which I reared *A. christophersi*. There were said to be fish in the tank, but they had no chance in the midst of vegetation so rank. So our unfortunate men had the enemy behind and before. The Inspector, Mr. Hoogwerf, on the other hand, lived on a low hill far from any standing water, and he had not found Diggi to be a feverish place at all, either for himself or his men.

Leaving Diggi and Mr. Pogson, I went on to Bamnia, regarded as one of the most feverish places on the frontier. Here I found rice-fields within one hundred yards of the lines, and though I got very few larvæ actually among the rice, they were plentiful in some moist ground adjoining. These were *A. christophersi*. On the other side of the lines there was a shady stream broken up into small, rocky pools, in which *Culex* and *Stegomyia* larvæ were plentiful and *Anopheles* not wanting. As I was clearing the bed of this stream with a gang of men the mosquitoes kept us very lively. Passing on to Kundal, another very unhealthy post, I found no rice-fields, but two streams within fifty yards of the lines. One was dry by this time, but the other was still running. It was a tiny rivulet, overhung by trees and bushes, the leaves falling from which choked the channel, so that the water filtered from one little pool to another. But the worst places, as usual, were the larger pools artificially formed by natives for watering their cattle and washing their clothes. Here I found larvæ abundantly and was able to give the Customs guards a good lesson. After an hour's work we had the satisfaction of seeing the little streamlet running swift and clear, and sweeping away all insect life in its course.

As a contrast to these places, I may mention Dhokarpa, a post which I was told had been very feverish two or three months ago, but was free now. Inquiring about water, I was told that there was none except one deep well. I found the bed of a stream, however, close to the lines, and asked when it had dried up. The answer was, at the end of the year. The strange thing is that probably every man on the frontier has had malarial fever many times and is liable to get it again at any season, yet they all recognise that certain places are "feverish" at certain times, and those times prove to be connected with the condition of a stream or a rice-field within a distance of two hundred yards. It seems to show that, even without treatment, a healthy native will soon shake off the effects of one infection, and that when malarial fever is severe and general, re-infection must be going on actively. The subject is one on which I have little right to put forward an opinion, but I state the impression produced on my mind by the facts which I observed.

Up to this time I had been travelling at a level of about 2,000 feet above the sea. I now descended the Ghauts and visited some posts on, or not far from, the coast, which are notoriously malarial about the end of the year. I was too late! I was told that the

¹ Hindustani mosquitoes.

fever had gone some time ago, and I searched in vain for the larvæ of any kind of *Anopheles*. There is no hot season crop of rice, and the streams were dry. In some there were still deep pools, full of dead leaves, but in these I found nothing. I came across one little rivulet, running sweetly and drawing a green line across the fields and along the side of the road, and here larvæ were plentiful, but happily we had no Customs post near to that place. All I saw in this tour confirmed me in a conclusion to which I came some time ago, that the one condition essential to make water habitable for *Anopheles* larvæ is that it should not be absolutely stagnant. They may be found in a filthy puddle or ditch if the tiniest stream of fresh water trickles into it, in a garden tub or cistern if it is freshened occasionally by rain, in a fountain if it plays sometimes, in a well if it is fed by a spring. And in rearing larvæ it is most important to change the water partly every day. With *Culex* larvæ there is no need to do this. The reason may lie in the nature of their food, but I do not think we really know yet what the food of *Anopheles* larvæ is. They are said to feed on *confervæ*, and I have often seen them browsing on those myself; but were they eating the *confervæ* or animal organisms on them? I am sure I have seen them eating the parasites off their own bodies and those of each other, and there is no doubt that they are very fond of animal food in other forms, for they always eat their own cast skins, and there is no situation where they may be found in greater numbers than in those collections of cast skins, dead midges, and other insect remains which may often be found on the surface of a pool. I have repeatedly watched them eating these remains. They feed almost incessantly as they float, fanning the water with their brushes and sending a current past their mouths, but the particles which they secure in this way may be either animal or vegetable. Some species also spend a good deal of time lying at the bottom and feeding on some *nescio quid* among the earth. In Bombay my experience has been that there is no sort of place in which they may be looked for with more certainty than pools formed by rain in freshly dug ground, which may help to explain the old theory about malaria being liberated from virgin soil. I think, however, that another reason why *Anopheles* larvæ cannot live in absolutely stagnant water may be that it favours the infusorian parasite to which they succumb so readily. The larvæ of *Culex* do not seem to be nearly so subject to this.

With respect to the practical results of my tour little can be said yet. I did not expect that anything I could do personally would effect much. My aim was to educate the more intelligent among our peons, and especially the petty officers, to take measures for their own protection. The results in this direction were distinctly encouraging. I found the men very ready to believe that fever was caused by the bites of mosquitoes, but none of them had the least idea that there was any connection between "fishes" which swim in water and "flies" which fly in air. When veritable mosquitoes were bred from the said fishes before their eyes all scepticism vanished and they entered keenly into schemes for the destruction of the miraculous vermin. The European Inspectors in

charge of the several beats also entered into the work with interest, as was to be expected. I did not encourage the use of kerosene oil. It is valuable as a last resort, but besides the objection that its effect is only temporary, it fouls the water and kills all the harmless and useful insects in it. I relied upon clearing the beds of the streams and connecting the detached pools, so as to induce a regular flow. Hollows in rocks were filled up, or emptied. Of course it was too late for any marked results to appear this season, but I hope that next year vigorous measures will be begun in time and maintained which will change the character of some of our most unhealthy posts. Where the focus of malaria is a rice field there is no hope, but I believe that it will be made a rule of the department soon, that lines are never to be built within a quarter of a mile of rice cultivation. Where there are lines so situated which cannot at once be removed, the experiment will be tried of destroying all intervening brushwood and grass.

A REPLY TO DR. BRADDON'S PAPER ON AN "UNDESCRIBED" HÆMATOZOON TO BE MET WITH IN THE MALAY PENINSULA.

By J. TERTIUS CLARKE.

District Surgeon, Lower Perak, Malay Peninsula.

IN the JOURNAL OF TROPICAL MEDICINE of September 16th, 1901, Dr. W. L. Braddon describes a new organism as the most frequent hæmatozoon to be met with in the Malay Peninsula, and has applied to it the term "mycoid." It is to be demonstrated by a process of wet staining, whereby a mixture of blood with a solution of methylene-blue in a 1 per cent. solution of potassium citrate is passed between a slide and cover-glass by capillary action.

In 152 out of 163 cases, Dr. Braddon says that this organism was certainly present, and in 98 was present alone, so that out of 163 cases of fever, in 11 only were the other ordinary organisms present alone, and it is to be gathered from this paper that he considers it the most frequent and, as the expression "severe fever" is used, the most important cause of fever in this country.

The appearances described are easy to see, in fact may be seen in almost every blood examined; it is not common to find a blood which does not exhibit them. My first few examinations were made upon blood from people who were fever free. I found it in a man who had not had fever for about two years, and was perfectly healthy; then in a woman who was menstruating, but who had never had fever; again, in a man who said that he had never had fever, and in another who had had fever for two days two years previously; in a cat apparently healthy, and it was found by a friend, Dr. Edgar, of Ipoh, in a dog and a frog. In these cases at least there was no connection between the so-called organisms and fever, but when I began to examine the blood of fever cases, my results as regards the finding of the ordinary forms differed considerably from those of Dr. Braddon; he failed to find the ordinary forms in 98 of his 163 cases, that is, in 60 per cent. I have examined 54 cases

of fever, and have found mycoids in 49 of these, but the ordinary forms in 43, or 79.6 per cent., two of my failures having had quinine. In many of these cases it has taken a considerable time to find the well-known organisms, partly due perhaps to lack of skill, but partly due to the frequent scarcity of the organism in the peripheral blood in some of the worst cases of fever. For instance, in one case of tertian fever, in which there were more mycoids than in any other case that I had examined up to that time, I had to examine five cover-glass preparations, three of them being stained, before I was able to find an organism; then, however, I found a few tertian rings; this difficulty I have met with more than once.

Dr. Braddon says that "the severity of the fever, and especially the resultant anæmia, have seemed to be in direct proportion to the numerical extent of the invasion."

I do not find that the numbers are in proportion to the severity of the fever. I do not find many in the early stages of fever, but very many in the malarial cachectic, and instead of speaking of the resultant anæmia, I should say that the numbers present are in proportion to the accompanying anæmia, of which it seems to be a denominator to a certain extent, rather than a cause. I have found them in the largest numbers in very anæmic cases which are not necessarily having fever; the blood containing the greatest numbers that I have come across was that of a person who was extremely anæmic, and whose stools contained ankylostomum ova. I have found many in the blood of a dysenteric patient who had been free from fever for a month; others showed large numbers who had had a long history of fever, but whose blood contained crescents, in whom, therefore, there is no need to ascribe the fever and anæmia to other than ordinary malarial parasites. In a case of spleno-medullary-leucocythæmia, sent to me by Dr. Edgar, there were many mycoids present, no fever, no history of fever, and no malarial organisms, but, as is usual in these cases, many nucleated red cells.

I have found mycoids also in people who on admission had fever and ordinary malarial organisms, whose fever was cured by quinine, but whose blood, after they had been free from fever for a considerable time (in one case a month), still showed many mycoids. From my results of the examination of the blood of man:—

(1) Mycoids are present in almost every blood examined, including that of the perfectly healthy.

(2) That in fever cases the ordinary organisms can almost, if not quite, always be found.

(3) That when present, the periodicity may be quotidian, tertian, or quartan, such periodicity seeming to depend on the malarial organism present, and to be unaffected by the mycoid.

(4) That perhaps most mycoids are to be found in cases of anæmia due to such causes as ankylostomum, or chronic dysentery, where, if present, fever is of secondary importance as a cause of the anæmia.

(5) That though the fever is cured by quinine the mycoid appearance is not removed.

My conclusion is therefore opposed to Dr. Braddon's, namely, that the mycoid appearance has nothing whatever to do with fever of any sort.

Most of the men with whom I have been able to

discuss this matter have regarded it not as an organism, but as an accident of staining—as an artifact; if so, it is an artifact more readily produced in some bloods than in others.

A reason in favour of its being an artifact is that the fluid used is not isotonic. In many specimens the hæmoglobin is dissolved out of the corpuscles, and as the mixture of blood and solution is not a perfect one, but varies in different parts of the preparation, it is to be expected that the hæmoglobin loosening effect will vary in different parts, and this is the case; in some parts there are many corpuscles freed of hæmoglobin, showing the appearances described particularly well, in other parts few of them have lost the hæmoglobin and here often not so many of the mycoids are to be found; this, however, does not prove that the appearance is artificial. I do not think that it is an organism; the motion described is the only point in favour of the view that it is an organism, and this, though I have failed to see it, is not conclusive. The numbers present in some bloods are so great that if it had any pathogenicity death might be expected at once, instead of large numbers being compatible with a considerable duration of life; in fact, instead of being pathogenic, I believe they are rather a sign of an attempt on the part of the organism to recuperate.

If not an organism, and if not an artifact, there still remains the possibility that it is a part of the blood corpuscles present under certain conditions.

Part II. of Dr. Braddon's paper, which was published in the JOURNAL OF TROPICAL MEDICINE, November 15th, 1901, in which he gives reasons for the belief that blood platelets are the precursors of the red cells, perhaps provides a clue as to what the mycoids may be. In it he deduces that the lymphocytes change by degrees to hyaline, then to transition, and then to polymorphonuclear cells; that the nuclei of these by subdivision or diffusion throughout the protoplasm give rise to these blood platelets, which in turn become converted into red corpuscles; the cytoplasm of the platelet being possibly thrown out or gradually absorbed by the plate of stroma; that is to say, he deduces that the red cell has originally a nuclear origin.

The nuclear constitution of the platelets has been shown by Lilienfeld, who by artificial pepsin-hydrochloric digestion was able to differentiate them into a pale homogeneous sphere and a nuclear mass; the fact that they are amphophilic may also point to a constitution partly nuclear. Lilienfeld is inclined to regard them, not as separate constituents of the blood, but as derivations of the nuclei of the leucocytes. Be that so, even if the red cells are not derived from the platelets they most probably have a nuclear origin, and if produced in this way, or in any other way, from a nucleus, it is possible that after being formed, the nuclear remains may not disappear all at once, but that in certain conditions, such as in rapid blood destruction or formation—to a certain extent correlatives—more young cells holding the last remains of the nuclear antecedent, perhaps the chromatic part, are sent into the blood-stream, and that these remains may be shown by a method by which the basophilic stain can get into the corpuscle without causing complete destruction of a network which is extremely delicate and already almost disintegrated. It is

possible that this last remnant of the creating nucleus, existing as it probably does in the central homogeneous cell protoplasm, on any process of fixing, is absorbed by, or intimately mixed with, the eosinophilic layer, so that it fails to take on the basic stain, being after its admixture too finely divided or otherwise changed to show its natural staining reaction, or perhaps even taking a faint neutral colour. The phenomena of polychromatism and of Plehn's dots may be explicable by the occurrence of the remains of a nucleus in the eosinophile layer; this, of course, is assuming that polychromatism is a condition of young cells and not a degenerative change. Many of the mycoids in human blood and in that of the embryo kitten, to be mentioned hereafter, resemble nuclei, though many of the denser ones are perhaps more filamentous in structure than any nucleus except those undergoing mitosis. The nuclear framework of a typical normoblast is described as consisting of a rather sharply defined network of chromatin having relatively wide intervening open spaces, so that the general appearance of the nucleus is not unlike that of a coarse net; this description is applicable to some of the mycoids and if one or two of these were present alone without the thread-like forms, they would most likely be called nucleated red-cells. In some very anæmic blood specimens which contained large numbers of mycoids, nucleated corpuscles were fairly common, as for example in the blood of the case of spleno-medullary leucocythemia before mentioned.

Believing that the mycoids are in reality the last remains of a nuclear progenitor, I turned my attention to very young blood, and examined several by the wet method described by Dr. Braddon. The first blood examined was that of an embryo crocodile which I got out of the shell—the hatching out would have taken place in perhaps five or six days. In this every red cell had fine blue-stained branched filaments radiating away from the nucleus, spreading through the entire cell, and forming, as it were, a fine network, on which the rest of the corpuscle seemed to be built up.

The outline of these cells, out of which the hæmoglobin had been dissolved, usually took the blue stain, suggesting that there is really a cell membrane, which is originally continuous with the nucleus by means of the filamentous processes, and I believe that the absence of the staining of this membrane will account for Dr. Braddon's description of some of these mycoids being free in the plasma. On taking blood from the unhatched egg of the fowl at various periods, a gradation was seen. In the blood of an egg which had only a very small streak of blood in it, the majority of the cells, on being treated by this method, were more like lymphocytes than red cells, the outer protoplasmic rim was more deeply stained than the nucleus; though the intensity of the stain varied, all the cells were circular. In the blood of a chicken about one week prior to being hatched, many of the cells were still circular; all had blue-stained filaments and dots, and some a blue halo of very fine filaments round the nucleus. On the following day there were fewer circular cells, but otherwise the appearances were similar, though less marked, and the lessening of the number of cells with filaments or with haloes round the nucleus went on until in an egg, which I expected

to hatch out on the following day there were very few and only indistinct filaments and no haloes. In the blood of the freshly hatched out chicken these mycoid filaments were difficult to find, in fact, the blood became like that of the adult frog, a few of the cells having blue-stained filaments. In none of these bloods did the filaments seem to join up the nucleus to an apparent limiting membrane, but there is an interval of about a week from about the eighth to the fifteenth day, during which I did not examine the blood of hatching eggs.

In all these bloods, whether of frog, chicken, or unhatched egg of chicken or crocodile, were the nucleus taken away these filaments would undoubtedly be described as Dr. Braddon's mycoid organism.

The blood of a kitten taken from the cat about one week prior to normal birth showed almost similar appearances to that of the unhatched eggs; very many blue-stained filaments and aggregations of filaments were seen, in fact, every gradation was present, from a few of the finest threads up to what was indistinguishable from a nucleus except that the filaments seemed rather more distinct. This blood was like the blood of the best specimens of mycoid containing blood that I have obtained from fever or anæmic patients.

On dry staining very many cells were amphophillic and some might even be described as basophillic; of the staining of the appearances described I am not certain, but I think that they can be seen to have taken on a neutral colour, so that in an eosinophillic mass the difference presented by filaments which cannot be seen by an ordinary $\frac{1}{8}$ -inch objective would naturally not be very marked, especially if there is a disintegration of them during the process of drying and fixing.

So from the result of my examinations of the blood of the unborn of an amphibian, a bird and a mammal, I should say that the mycoid filaments are the remains of the branching in some cases, or the breaking up in others of the original nucleus; that red cells have all a nuclear origin and an undoubted cell membrane, which is originally connected with the nucleus, but perhaps ultimately separated by the growth of the cell, during which process in mammals the remains of the nucleus is completely absorbed.

In one of the fever cases in which I failed to find mycoids the man was moribund, and it is possible that in him the formation of red cells had ceased, in which case, if my theory that this mycoid appearance is only to be found in young cells, be correct, it is not to be expected that they would be found. In another man they were not found, he had no fever, was healthy, but below par.

If, as Dr. Braddon alleges, this mycoid is the most frequent cause of "severe fever" in this country, it will not be of much use to expect Government or private individuals to use measures for the prevention of the fevers caused by the parasites whose life history is known, until the life history of this is known also. If, on the other hand, this mycoid is not an organism, but a purely physiological phenomenon, as I believe I have shown, the statements made by Dr. Braddon should not be allowed to delay the prosecution of those means which have been shown to be efficacious in the prevention of fevers.

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THE

Journal of Tropical Medicine

NOVEMBER 1, 1902.

A RECENT ADDITION TO TROPICAL PARASITOLOGY.

THE case of trypanosoma which we report in this issue of the Journal opens up a new field for investigation and elucidation. In the JOURNAL OF TROPICAL MEDICINE for September 1st, 1902, we published an article by Dr. R. M. Forde, on "Trypanosoma found in the blood of a European," and gave an illustration of the parasite. The patient from whose blood this parasite was obtained was shown in Liverpool on August 1st, 1902, to the members of the Section of Tropical Diseases who visited Liverpool during the meeting of the British Medical Association at Manchester. The disease was at the time regarded rather in the light of a curiosity, but the discovery by Dr. Daniels, Superintendent of the London School of Tropical Medicine, of the same parasite in a patient in whom the disease was suspected by Dr. Manson, alters the whole aspect. What was then regarded as a curiosity has by the dis-

covery of a second case brought us face to face with a possibly fairly widely, or even widely, distributed disease. Whatever the geographical distribution may be, the fact remains that the trypanosoma in the blood occasions a definite ailment, associated with pronounced clinical signs and symptoms. A new clinical fact is before us, a new fact in parasitology. In West Africa and in the Upper Congo, at all events the districts from whence Dr. Forde's and Dr. Manson's cases were obtained, the possibility of encountering the disease must be remembered, and it is possible that far beyond these areas the parasite and its attendant clinical features may be met with in other districts and in other continents.

The discovery is another step in the disentanglement of tropical ailments. The enormous mass of diseases grouped together, merely from the fact that they were tropical, is being reduced bit by bit, as now this investigator and that observer throws light on this or that ailment, which had previously been believed to belong to the mass—tropical. Malaria served for many years as a cloak to render our ignorance obscure, yet from its colossal mass many diseases have been abstracted, including such well-established specific ailments as beri-beri, kala-azar, &c., and even such distinct diseases as hepatic abscess and dysentery, and the many febrile states met with in filariasis, siriasis, &c. Many other ailments have been dissociated from the chaotic mass and it is satisfactory to know that work in this direction is advancing. The latest discovery—that of trypanosoma in the blood of man—is but another evidence that our knowledge of tropical diseases is progressing, and that the scientific workers of to-day are establishing a basis for future investigation which will redound to their credit for all time.

A CASE OF TRYPANOSOMA IN A EUROPEAN AT THE SEAMEN'S HOSPITAL, ROYAL ALBERT DOCKS, LONDON.

Under the care of Dr. MANSON, C.M.G., F.R.S.

DURING the meeting of the British Medical Association in Manchester, last August, Dr. Manson had the opportunity of seeing a patient in whose blood trypanosomes had been found by Dr. R. M. Forde. He was struck by the peculiar clinical facies, the

chronic irregular fever, the enlargement of the spleen, the oedema of the face particularly, and the very well-marked erythema multiforme scattered over the trunk and limbs.

A month ago Dr. Habershon sent a patient to Dr. Manson for an opinion. The patient was the wife of a missionary on the Upper Congo, where she had resided for about a year. She stated that at first her health was fairly satisfactory with the exception of a few slight fevers, but latterly, the fever becoming more persistent and her general health impaired, she was invalided home, and since last December resided in England. Notwithstanding liberal drugging with quinine and arsenic, her condition was not improved. On examining her, Dr. Manson recognised the same grouping of symptoms that he had seen in Dr. Forde's patient. The fever, the enlargement of the spleen, the oedema of the face, not very marked but at the same time distinct, and the multiform erythema, made him suspect the nature of the case. He persuaded the lady to enter the Tropical wards of the Seamen's Hospital, Royal Albert Docks, so as to be near the London School of Tropical Medicine, where systematic examinations of her blood were made daily for a fortnight. No trypanosomes being discovered, arrangements were being made to test the tentative diagnosis by injection of the patient's blood into several of the lower animals. Before these arrangements were completed, Dr. Daniels, Superintendent of the School, while counting the leucocytes in the blood of the patient, came across an unquestionable trypanosome. On the examination being repeated, other specimens of the parasite were readily discovered. For some unknown reason the parasite must have been latent during the first part of the patient's residence in hospital, or so very scanty in the peripheral circulation that they were missed, although diligently sought for. It would appear, therefore, that Dr. Forde's case is no mere pathological curiosity, and that in future the pathologist has to reckon with a new disease-germ in man, with a geographical range probably involving the whole of Tropical Africa. And if Africa, why not Tropical Asia and America? Possibly in this parasite we have an explanation of some of the anomalous chronic fevers, such as kala-azar, which have hitherto defied investigation in tropical countries. We trust that those who have the opportunity will take advantage of it to determine the extent and practical importance of this new parasite; and that they will examine it in this light with the corresponding diseases of the lower animals, especially of surra, nagana and mal de caderas.

The case will be published in full detail in an early issue.

LABORATORIES IN THE PHILIPPINES.—A large central institution, in which laboratory work shall be done for all Government departments requiring scientific assistance, is to be established in the Philippines. There will be rooms for bacteriological diagnosis, for pharmacology, pathology, food analysis, &c. Altogether the building will contain sixty rooms. A serum institute also, with every requisite in the way of serum packing rooms, serum kitchen and crematory, is to be erected.

British Medical Association.

BERI-BERI.

By L. W. SAMBON, M.D. (Naples).

London School of Tropical Medicine.

IN the discussion on Dr. Manson's paper, Dr. Sambon said: Dr. Travers' observations and experiments are certainly interesting, but I fail to see how they can sweep away the enormous amount of evidence brought forward against rice not only by Baron Saneyoski, F.R.C.S.Eng., in his admirable report on the experience of the army and navy of Japan, but by the great majority of physicians of all times who studied the disease in China, Japan, the Dutch Indies, and Brazil. It is, of course, impossible for me to discuss Dr. Travers' experiments on the data furnished by Dr. Manson, but I will just point out that the rice, though supplied by the same contractor, was not the same in both gaols, because it was bought from several Penang merchants, who themselves collected it from many districts in separate bags. Then again it was only for a very short period (from October 1st to December 14th, 1895) that rice stored and cooked in the Pudooh Gaol was supplied to the inmates of the Old Gaol, and this whilst the disease was decreasing in the Pudooh Gaol.

Certainly, I do not think that rice causes beri-beri because of its deficiency in mineral and nitrogenous matters, but I am quite prepared to admit that it may be related to beri-beri in the same way as we now believe pellagra to be related to maize, or, in other words, that rice may become a vehicle of the beri-beri infection. Rice is used as a staple food over a far wider area than that in which beri-beri prevails, but the cause of the disease is not the rice itself, but perhaps some micro-organism which, in certain places and under certain conditions, may be associated with rice and possibly with other grain.

Within the endemic centres of beri-beri it has been frequently noticed that the disease is most common in communities supplied with rice of inferior quality, or with rice which has been carelessly prepared and badly stored. Paddy keeps sound for years, but the grain deprived of its pericarp is soon damaged by vegetable parasites. Dr. C. Eijkman, from statistics applying to 280,000 prisoners, showed that in the prisons of Java the proportion of beri-beri cases is 1 to 39 in convicts fed on decorticated rice (white rice), whilst it is 1 to 10,000 in convicts who consume the grain half peeled, that is to say, deprived of pericarp but still enclosed in its perisperm (red rice).

Besides fungi, there are numerous insects and mites that live on stored rice, and the granaries are constantly visited by mice and rats. Most interesting associations have been established between all these plants and animals that gather on rice. For instance, *Pintus latro* is a small beetle that feeds on rice in winter, its larva inhabits the excrements of rats. If rats were liable to beri-beri, as Lacerda suspected, the specific microbe might be conveyed in their excreta. I mention these facts merely to show how

complex might be the connection between rice and beri-beri.

Dr. Manson believes that beri-beri is due to a toxin prepared by a germ outside the human body, and possibly inhaled or absorbed through the skin. He bases his theory chiefly on analogy with the multiple peripheral neuritis of Europe, which is attributed to alcohol, and is, therefore, called "alcoholic paralysis." I doubt very much whether alcohol alone can produce peripheral neuritis. In this very town, two years ago, when there was quite an epidemic of multiple neuritis, Dr. E. S. Reynolds attributed the disease to arsenical poisoning, and stated how he had doubted for years whether ethylic alcohol caused neuritis. He had noticed that the disease occurred only in beer drinkers, and found that the beer contained arsenic. But if Dr. Reynolds' assumption can explain satisfactorily the Manchester epidemic of 1900, what about the outbreaks of multiple neuritis which occurred more than a century ago when beer was manufactured without the glucoses and inverts of modern chemistry? Dr. Lettsom, in his "History of Hard Drinking," published in 1789, describes multiple neuritis quite clearly; and multiple neuritis has occurred every year in Manchester, like other diseases of microbic origin, varying in prevalence from year to year in connection with as yet undetermined surrounding conditions; sparing some districts entirely, weighing heavily on others; attacking children as well as adults, and abstemious persons as well as drunkards.

If beer drinking caused the Manchester epidemic of 1900, what about the outbreaks of multiple neuritis which occasionally take place in Italy, in France, and in other countries where beer is never or hardly ever consumed; and if the disease is due to arsenic, which may be contained in wine as well as in beer, how shall we explain the immunity of the arsenophagous mountaineers of Austria and Styria, and that of the many patients, suffering from epilepsy, chorea, or anæmia, who are given excessive doses of arsenic?

I am inclined to believe that all forms of multiple peripheral neuritis, both in Europe and in the East, are the outcome of specific infection. It is quite possible, however, that the administration of alcohol or arsenic may favour the development of the microorganisms of multiple neuritis. These parasites, like *aspergillus glaucus*, *mucor mucedo*, and other arseniobacteria, may have a special affinity for arsenic and other poisons. We know that arterio-sclerosis and gout are especially common amongst lead workers, and we know that quinine may provoke relapses of blackwater fever; but in these and other similar instances the specific cause of the disease must also necessarily be present. Numerous experiments by Abbot, Delcarde, Goldberg, Koch, and others have proved that the administration of alcohol to animals before infection greatly increases their susceptibility and destroys both natural and artificial immunity. Multiple neuritis occurs very frequently in the convalescent stage of the acute infections, such as typhoid fever, small-pox, influenza, pneumonia and diphtheria, or during the course of chronic diseases, such

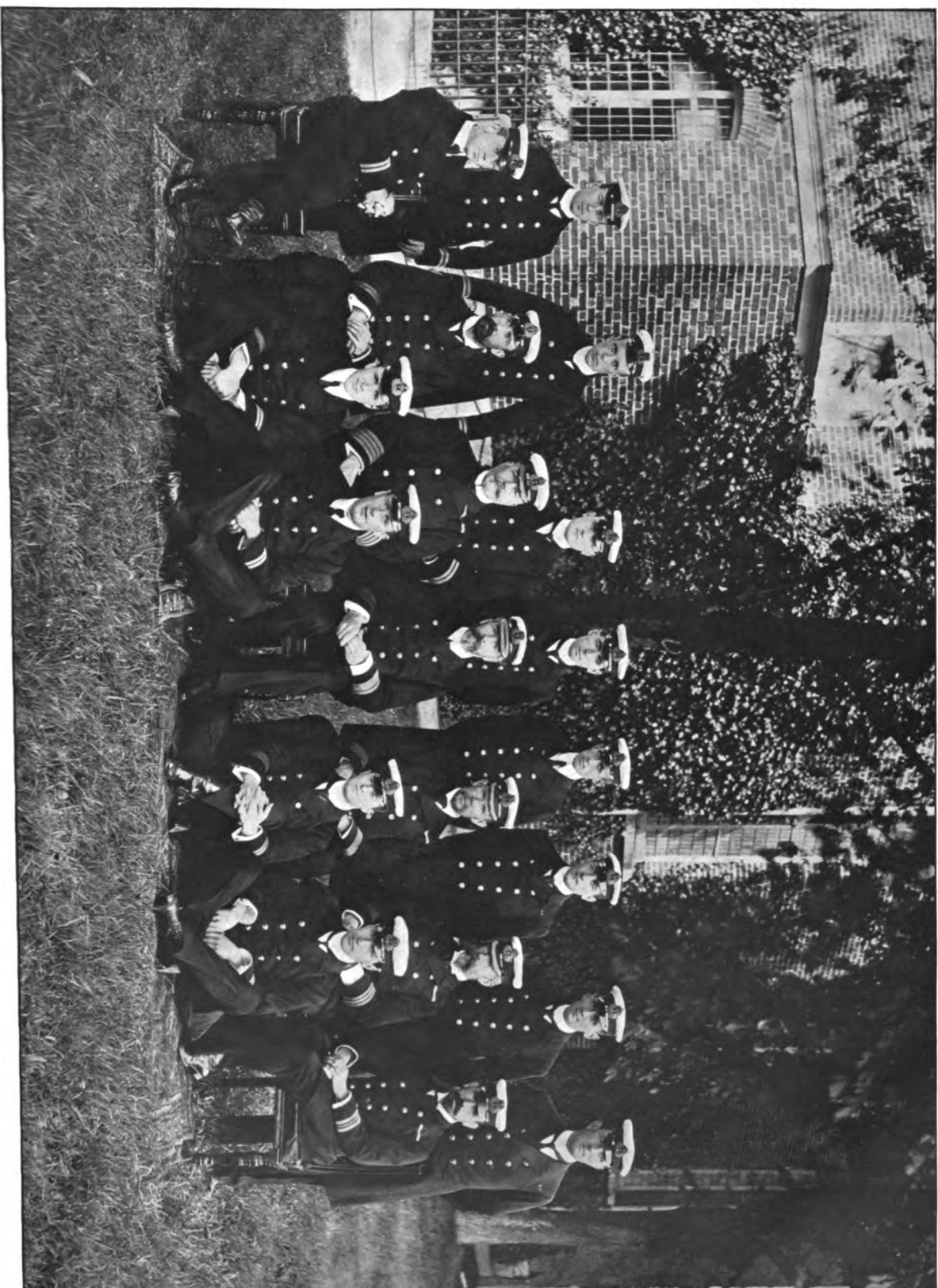
as leprosy, syphilis, and tuberculosis; or, again, in the cachectic stage of the malignant neoplasia and of the intermittent fevers. It would seem, therefore, that, like alcohol, arsenic, lead, or carbon bisulphide, the microbic poisons of certain diseases were also able to prepare a substratum favourable to the development of the agent which causes multiple neuritis.

I believe that the specific agent of beri-beri lives within the patient's body, and attacks the peripheral nerves. I base this assumption on the clinical symptoms and anatomical data of the disease, which show, most strikingly, a different distribution of the pathogenic agency in different patients and in the same patient at different stages. Moreover, the same diseased nerve may show simultaneously normal fibres, degenerated fibres, and regenerating fibres, and the degenerative changes of the medullary sheath and axis-cylinder do not affect the nerve-fibre at once throughout its whole length, but proceed slowly from the periphery to the centre. Another point in favour of my supposition is afforded by the fact that beri-beri may remain latent for long periods within the system. This latency is evidenced by the length of the incubation period, which at times seems to extend over several months, and by the frequency of relapses in those who have contracted the disease. This latency of beri-beri has not been sufficiently appreciated. It accounts, I think, for the numerous outbreaks of the disease amongst gangs of coolies out at sea or landed in places in which beri-beri did not previously exist, and it explains the strict limitation of the infection to such gangs which has been so frequently observed, because undoubtedly beri-beri cannot spread any more than malaria in the absence of its peculiar agent of propagation.

In discussing the etiology of beri-beri we should not forget that although the primary cause of the disease is still unknown, there are many important epidemiological facts which have been thoroughly ascertained. Thus we know that the prevalence of beri-beri within its endemic area varies greatly from year to year. We know that the disease has a seasonal prevalence most noticeable in those regions which have marked seasons, and we know that its prevalence, like that of the intermittent fevers, is favoured by high temperature and an abundant rainfall. Then, again, we know that beri-beri is more common in certain races, not on account of a racial susceptibility, which does not exist as an ethnic character in any race, but because the stricken races inhabit beri-beri centres, or because their customs and habits bring them into closer touch with the cause of infection. Beri-beri is more prevalent amongst men than women, and amongst adults than children, but these facts only prove a greater exposure of the adult males, and not sex or age immunity. In fact, we know of several outbreaks limited to women or very young children, and we know of cases in suckling infants and in men over 70 years of age. Occupation is another condition which must be taken into account. Beri-beri may attack men of any class or occupation, but it is chiefly the disease of the common labourer. Beri-beri is known to attack robust, healthy men in the prime of life, but it has

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STAFF OF HASLAR HOSPITAL AND THE JUNIOR OFFICERS ATTENDING THE COURSE OF INSTRUCTION DURING THE TERM ENDING OCTOBER, 1902.



Surgeon R. L. Jones Surgeon W. B. Maurice Surgeon C. A. G. Phipps* Surgeon R. H. Atkins Surgeon W. R. Harrison Surgeon F. E. Bolton Surgeon G. K. Bushe Surgeon H. C. Arthoon
 (Hospital Staff) Dep.-Jasp.-General James Porter, C.B. Insp.-General R. W. Coppinger Fleet-Surgeon Gilbert Kirket Fleet-Surgeon W. Tail Staff-Surgeon P. W. Bassett-Smith
 Surgeon G. T. Bishop Surgeon W. G. Edwards Surgeon J. A. Thompson

a remarkable predilection for syphilitic and tuberculous patients; for those recovering from small-pox, scarlet fever, measles, typhoid fever, or dysentery; for women in the puerperal state, and for those suffering from eye diseases, from ulcers of the extremities, or from surgical wounds.

We are absolutely ignorant of the way in which the beri-beri infection is carried from place to place and from man to man. We know that usually the disease does not spread in hospitals among other patients when cases of beri-beri are admitted, and that it does not attack nurses or visitors. On the other hand, we know that the disease has been carried to new countries. Thus, it was introduced into New Caledonia in March, 1891, by a shipload of Tonkinese and Annamese prisoners. It broke out first amongst the prisoners and caused many deaths. After a time the disease spread, and in some instances proved fatal to certain natives, Kanakas, who had become associated with the Asiatics. Then, again, beri-beri is essentially a disease of collective dwellings such as gaols, schools, asylums, hospitals, barracks, and ships, a fact which at first sight might suggest contagion; and indeed overcrowding has been looked upon as one of the chief causes of beri-beri, but overcrowding has been removed in prisons and asylums without any definite improvement. Sometimes beri-beri has broken out in old insanitary buildings and in wretched mercantile ships, but just as numerous are the examples of outbreaks in new buildings and modern battleships apparently under the best hygienic conditions. In many cases the disease has persisted after the temporary removal not only of those attacked, but of all inmates or crews and after the most rigorous disinfection.

The persistency of the disease in collective dwellings notwithstanding the most careful disinfection, suggests the idea that beri-beri might be a place disease. But there are facts which oppose this theory. For instance, in April, 1894, 250 Japanese coolies were imported to a sugar plantation at Labosa in Fiji. Beri-beri had never occurred before in this colony. The Japanese were placed on an outlying plantation where Indian coolies were also employed. The dwelling houses were especially built for them. One month after arrival, beri-beri broke out, and by the second week in November 219 out of the 250 had contracted the disease. In February, 1895, the 181 survivors were sent back to Japan. Many of these Japanese coolies had, previous to immigration, suffered from beri-beri. On the repatriation of the Japanese, their houses, after disinfection, were divided into separate compartments and occupied by Indians, yet no cases of beri-beri followed.

To explain the peculiar features of the endemicity of beri-beri some authors have suggested that possibly the disease agent is spread by insects or other animals living in contact with man. Some have incriminated fleas, others lice, others mosquitoes. Van der Scheer accused the common cockroach (*Blatta orientalis*), which is found in all parts of the tropics, dwells in houses and ships, visits the larder and the latrine.

Beri-beri is a disease of the utmost scientific interest

and of great economic importance, and therefore it was very properly chosen as a subject for discussion; but I fear that academic disquisition will not advance very much our knowledge of its etiology. What is needed is thorough local investigation by competent men.

Without a definite knowledge of the cause of beri-beri it is, of course, impossible to formulate strict prophylactic rules. However, I think we might suggest, on the experience so far collected, the following provisional measures:—

(1) Men who have recently suffered from beri-beri should not be allowed to join coolie gangs, because they would most probably suffer from a relapse of the disease and possibly become a source of infection to others.

(2) Patients suffering from beri-beri should be isolated in beri-beri countries, because, under certain as yet undetermined conditions, they are undoubtedly a cause of further infection.

(3) Open wounds, ulcers, and abrasions of any kind should be most carefully cleansed and dressed in those who are obliged to associate with beri-beri patients.

(4) The diet supplied to coolie gangs and to the inmates of collective dwellings should be varied and rich in nitrogenous ingredients. The rice should be of good quality, recently decorticated, properly stored, carefully washed, and thoroughly cooked.

Whatever may be the cause of beri-beri, we cannot deny that diet seems to have a potent influence on its prevalence. Takaki's dietetic reforms in the Japanese navy to overcome beri-beri have proved as great a triumph as those introduced by Blane into the English navy in 1795 to combat scurvy—a disease in many ways comparable to beri-beri.

ENTERIC FEVER IN SIERRA LEONE—NOT YET ENDEMIC?

WITH A NOTE ON A CASE OF INFECTION THREE MONTHS AFTER CONTACT.

By MAJOR F. SMITH, R.A.M.C.

Sierra Leone.

ENTERIC fever has not been generally regarded as a Sierra Leone disease, and, as a matter of fact, it is very rare indeed. The object of this paper is to record the fact that enteric is among us, to moot the question as to whether it is a new thing or not, and to point out a danger which overhangs us in the shape of an extension of the disease. Our insanitary surroundings are likely to conduce to its establishment in Freetown. The city contains some 40,000 inhabitants spread over a large area, and the place is honeycombed with unsteined wells and cess-pits, the latter generally at a higher level than the former, and within twelve yards' distance. A great part of the inhabitants do not yet use any common water supply. These conditions are more likely to bring about an endemic state than to give rise to a sudden extensive epidemic.

It seems an extraordinary thing that enteric has

not been more common in Sierra Leone considering that there is nothing to prevent it from travelling from the north of Africa, where it is common, or from South Africa, where it has played such havoc with our soldiers.

Malaria has, however, been hitherto regarded as almost our only fever, and the facts seem to justify this view. The absence of records of enteric cannot be looked upon as being to any great extent due to cases having escaped the notice of medical men. We have had skilful physicians here since the early part of last century, men with experience of diseases of other countries, and they are not likely to have all of them overlooked the existence of a malady having such marked characteristics. No doubt many like myself have been on the look-out for it.

The following is a list of all the causes and suspected cases I have heard of:—

LIST OF CASES OF ENTERIC FEVER AND SUSPECTED CASES IN SIERRA LEONE FROM REMOTE TIMES TO MAY, 1902.

| No. of Case | Date or Approximate Date | Remarks |
|-------------|--------------------------|---|
| 1 | 1892 .. | Negro soldier of the West India Regiment at Tower Hill. Died three weeks after arrival in the country. Probably contracted disease elsewhere. |
| 2 | Dec., 1898 | Case not returned as enteric, but came under my care towards end of attack, and I suspected enteric (European). |
| 3 | Late in 1899 | Native negro soldier reported by Dr. Horrocks, and verified <i>post mortem</i> at Panguma, far away in the hinterland. |
| 4 | 1900 .. | Case landed from a man-of-war. Died a few days later in the Colonial Nursing Home. |
| 5 & 6 | | Dr. Renner informs me that he had two suspicious cases among the European residents. |
| 7 | 2nd quarter of 1901 | A West Indian negro woman in Freetown. Two attacks of hæmorrhage. Brought to my notice by Dr. Latchmore. |
| 8 | 2nd quarter of 1901 | West Indian negro soldier in Tower Hill Barracks. Verified at necropsy. |
| 9 | Dec., 1901 | A suspected case in a Freetown native. |
| 10 | Jan., 1902 | Ditto. |
| 11 | Feb., " | Three native children living in one house at Freetown. |
| 12 | " " | |
| 13 | " " | |

The diagnosis in Cases 11 and 12 is strengthened by the fact that the dried blood examined by Professor A. E. Wright in the laboratory of the Army Medical School at Netley gave positive reaction in the agglutination test.

Eight of the 13 cases, namely, Nos. 2 and 7 to 13 came under my personal observation. Though there is no definite evidence of any connection of these cases one with another, the sequence of occurrence is such as to suggest relationship between Nos. 2 and 3, also between No. 7 and all those which followed except No. 8.

Case No. 2 was in a man who had recently arrived from Egypt, where enteric was rife. He was taken ill almost immediately after arrival in this country, and while on service in the hinterland. He was brought down through Panguma, and stayed there

two days in quarters in the barrack enclosure, in which Case No. 3 occurred a few months later.

Case No. 7 is dealt with below, but of those which followed No. 8 was in a soldier in isolated barracks on a hill, and was most likely connected with the arrival early in 1901 of some comrades who came from St. Helena in the same ship which brought the case No. 7.

The remainder occurred on the same side of the town as No. 7, but the houses were by no means near and there was no common water supply. It is probable, though, that the clothing was washed in the same stream. Dirty clothes in Sierra Leone are "taken to the brook" and washed in the pools which in the dry season are more or less stagnant, but communicate by rivulets of running water—hot water is never used by our washerwomen. These cases, might, of course, have arisen out of Nos. 2, 4, 5 or 6; or, again, out of unrecognised cases from the hinterland.

Case No. 7 bears on the question as to whether or no enteric fever is always waterborne. The woman was in St. Helena with her husband; she came to Sierra Leone in the middle of January on a transport which had carried soldiers and Boer prisoners from South Africa. Towards the end of the following April she developed enteric fever. Her husband, who came with her, had not been ill, neither had her only child or any one connected with the family. The woman was in lodgings in the town, where, as before stated, the disease is almost unknown. No local source of infection could be discovered.

There is little doubt that in this case the germs of infection came from St. Helena or the transport. The assumption, then, is that the infection was carried about on the person or belongings of the woman or some member of her family for some weeks before it found its way into her system.

It will be gathered from the remarks on the other cases that there is no certainty about the origin of this case No. 7; but the rarity of the disease in Sierra Leone, and the absence of evidence of other cases occurring in or near the house before this West Indian woman was attacked, together with the important point that she had recently come from an infected place, is strongly in favour of the theory that she brought the bacillus with her.

The serious fact in any case is that we have enteric fever in Sierra Leone; and it is well that all should know it in good time. Our climate is bad enough already, without the addition of typhoid to our burdens.

STATISTICS OF THE BLOOD EXAMINATION IN CASES OF MALARIA IN CYPRUS DURING A PERIOD OF TWELVE MONTHS.

By GEORGE A. WILLIAMSON, M.A., M.D. Aberd.,
Colonial Medical Service, Cyprus.

"MALARIA is therefore a local phenomenon which must be studied on the spot, and the data gathered in any particular territory cannot be generalised or applied to all countries." Such is the opinion expressed by the Italian authority, Professor Calli.¹

If, however, there were available statistics for each territory or locality, a general malaria map might be constructed, which, besides being deeply interesting as showing the types of fever peculiar to the several places, would be of great use for commercial and other bodies in enabling them to more accurately gauge the risks to which their employes in such regions would be exposed from this, the commonest disease of hot climates.

One hears frequently the terms coast fever, West Indian fever, Roman fever, Cyprus fever, &c., employed, as though they were entirely separate diseases instead of being, as they are, malarial fevers, the difference between them depending on the variety of malarial parasite causing the greatest number of cases or the most striking class of cases; thus the parasite occurring almost exclusively in malaria in some parts of Sierra Leone was found to be the hæmomenas præcose. Statistics such as those following, if available for our different colonies, would help to a more intelligent understanding of the real meaning of such terms as those mentioned.

FREQUENCY OF THE PARASITES.

These statistics are the results of careful microscopical examination of the blood in all cases of illness where the symptoms at all resembled those of malaria, and extend over a period dating from February 1st, 1901, to January 31st, 1902.

TABLE I.

| | |
|--|-----|
| Number of cases examined for parasites .. | 729 |
| Number of cases diagnosed as malaria .. | 508 |
| Number of cases in which parasites were found .. | 470 |
| Percentage of the cases diagnosed as malaria, in which parasites were found, equals 94.4 per cent. | |

Davidson² says that Laveran in Algeria found parasites in 432 out of 480 cases examined, and that Osler, Councilman, and James in the United States found them in from 90 to 100 per cent. of their cases—results much the same as those on which the present remarks are based.

THE MALARIAL SEASON.

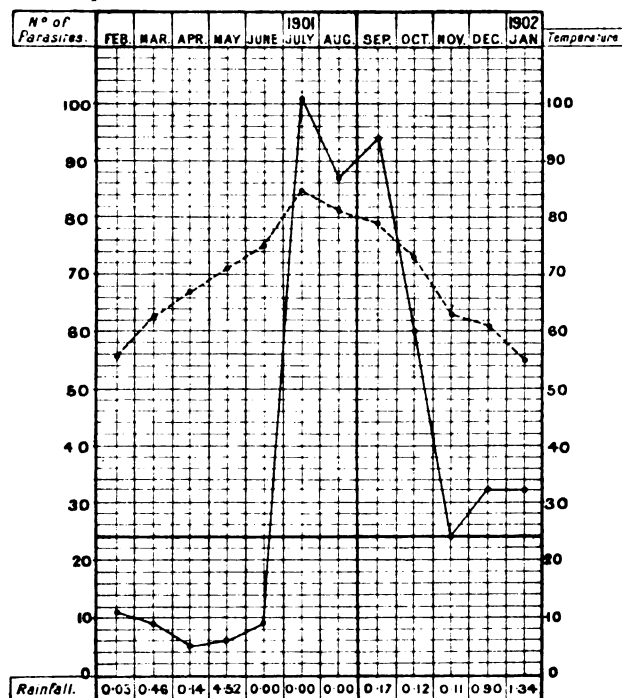
In Table II are given, for the different months, figures showing the number of cases in which malarial parasites were found, and from this it is at once evident that the malarial season is the second half of the year.

TABLE II.—Showing for the Several Months the Number of Cases in which Malarial Parasites were Found.

| Date. | Cases. |
|-------------------------|--------|
| February, 1901 | 11 |
| March, 1901 | 9 |
| April, 1901 | 5 |
| May, 1901 | 6 |
| June, 1901 | 9 |
| July, 1901 | 101 |
| August, 1901 | 87 |
| September, 1901 | 94 |
| October, 1901 | 60 |
| November, 1901 | 24 |
| December, 1901 | 32 |
| January, 1902 | 32 |
| | 470 |

The same figures are used in the construction of chart A, which shows in a most striking way the seasonal prevalence. The greatest number of cases

occur in July, and from then on to November there is a gradual drop, followed, strange to say, by a slight rise in December and January. This rise is not usual in Cyprus, and is perhaps to be ascribed to a downpour of rain at the end of September, followed by a long period of dry weather, thus affording an opportunity for the breeding of mosquitoes in the pools left; and, in fact, the number of mosquitoes to be found through the winter up to January was very generally commented on in the district as being quite unusual; it is generally held here that the heavy rains, which as a rule fall in October and November, scour out the pools and wash away the larvæ, but in 1901 there was no continuance of heavy rains to act in this way, and the one downpour in the end of September merely supplied the necessary pools for the development of the mosquitoes.



A.—Chart showing (1) number of malarial parasites, (2) mean atmospheric temperature, and (3) rainfall, for twelve months, from February, 1901, to January, 1902. Black line, number of parasites; dotted lines, temperature.

Chart A shows the rainfall in inches during the twelve months, and it will be seen that the very small amount of rain falling on the earth makes any connection between annual rainfall and malaria incidence difficult to show. The average rainfall for Cyprus is about 12 inches, but in Larnaca during the twelve months in question it was only 7.8 inch.

So far, the following propositions may be hazarded:

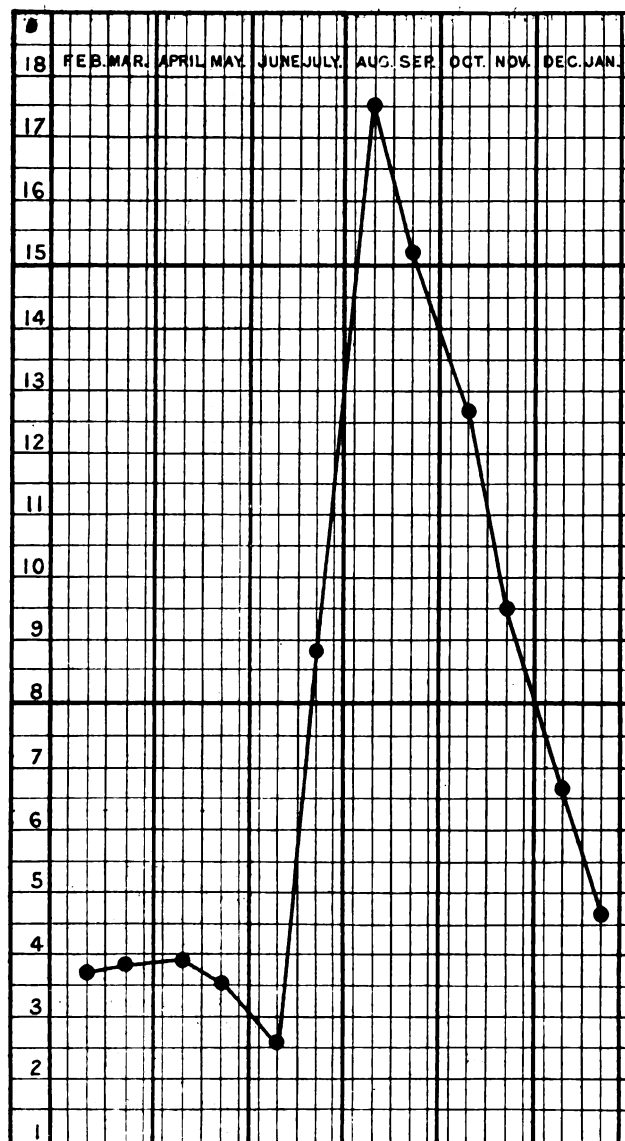
(1) That in years with a small winter, and more especially spring, rainfall, the summer malaria prevalence will be low.

(2) That in years with a great winter, and more especially spring, rainfall, the summer malaria prevalence will be high.

(3) That with frequent and severe autumn and early winter rain, the winter malaria prevalence will be very low indeed.

(4) That with late summer rain followed by autumn and early winter drought, the winter malaria prevalence will be, for the season, high.

During this present summer the mosquitoes have so far been much fewer than usual, apparently owing to the absence of spring rain to form breeding pools, and malaria has been later than usual in appearing in force.



B.—Chart, compiled from the published statistics of Professor Celli, showing the number of cases of malarial fever observed monthly, in the hospitals of Rome, for the years 1864, 1865, 1873, 1874, 1877, 1878, 1892, 1893, 1894, 1895, 1896, 1897, 1898. Each division represents 1,000 cases, and the total number of cases for the whole period amounts to 93,000.

COMPARISON WITH CELLI'S STATISTICS.

Chart B is compiled from figures given by Celli.⁸ The general curve is very much the same as that for Cyprus for 1901; in both the fact that it is the second half of the year that is malarial rather than the first is clearly brought out, and this concurrence, con-

sidering the geographical position and meteorological conditions, is what might be looked for.

MALARIAL PREVALENCE AND ATMOSPHERIC TEMPERATURE.

Reference to Chart A shows that there is a certain likeness in the two curves, that of malaria and of the mean monthly temperature. The three months when most fever cases occurred were July, August, and September, and these were the three hottest months also, and were the three months during which (with the exception of a downpour on September 30th) no rain fell. These circumstances go to show the truth of Celli's remark that malaria is a local phenomenon, as will be more clearly brought out if we compare these results with those given in the Report of the Malaria Expedition to West Africa,⁴ with reference to malarial admissions among the troops in Sierra Leone; there the greatest number of admissions were stated to occur during June, July, and August, the three rainiest months being July, August and September, these months being also the coolest.

In this connection it is interesting to note that the mosquitoes do not here hatch out in numbers until April, and it has struck me that *Anopheles* hatch out at a slightly later date than *Culex*—this fact, dependent probably on atmospheric temperature, would tend to explain the seasonal incidence of malaria.⁵ The fever season in Algeria appears to be much the same as in Cyprus, occurring as it does during July, August, September, which, with June, are also the months with least rainfall.

SEASONAL PREVALENCE OF THE DIFFERENT VARIETIES OF MALARIA.

Chart C gives the curve for tertian, quartan and æstivo-autumnal fevers respectively, and is compiled from the same figures as Chart A, the different varieties of parasites found being separated.

From this the tertian is seen to be an earlier form than æstivo-autumnal, its greatest prevalence occurring in July, whereas the greatest prevalence of æstivo-autumnal occurs in September—in fact, tertian is an early summer and æstivo-autumnal a late summer fever—both, however, declining rapidly from September onwards, a slight rise of æstivo-autumnal making its appearance in January, this slight rise is evident in both Charts A and C, and an attempt at its explanation has already been given.

The cases of quartan are so few in comparison to the others that the curve is of little value—it would, however, point to quartan being here, as elsewhere, a cold weather form of malaria—it is to be noted that cases were met with throughout the whole year.

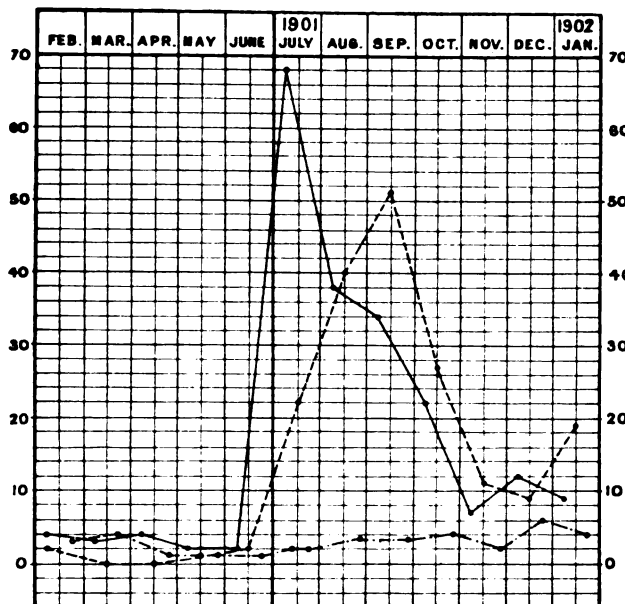
The percentage of the different varieties, with reference to the total number found, works out as follows:—

| | | | | | | |
|-----------------|----|----|----|----|----|--------|
| Tertian | .. | .. | .. | .. | .. | 48.46 |
| Quartan | .. | .. | .. | .. | .. | 8.03 |
| Æstivo-autumnal | .. | .. | .. | .. | .. | 43.50 |
| | | | | | | 100.00 |

This percentage prevalence very closely agrees with that given by Davidson⁶ as applying to Vienna:

| No. of Cases | Quartan | Tertian | Quotidian | Irregular |
|--------------|---------|---------|-----------|-----------|
| 3,126 | 7.8 | 47.8 | 41.3 | 3.0 |

His figures for India, Algeria and Sweden vary very largely from these, showing a decrease in the quartan and tertian, and an increase in the quotidian as the colder climates are changed for the warmer.



C—Chart showing, monthly, the number of parasites of (1) tertian, (2) quartan, and (3) æstivo-autumnal malarial fevers, from February, 1901, to January, 1902. Black line, tertian; dash and dotted lines, quartan; dash line, æstivo-autumnal.

MULTIPLE INFECTION.

The following table (No. III.) shows the total number of cases of tertian and quartan during the twelve months, and also the proportion of cases that were of double or triple infection. From this it is evident that the multiple infection in tertian fever is most marked during the season of greatest malaria prevalence, which, as we have seen, is also the season of greatest heat.

With regard to quartan the frequency of triple quartan (which without microscopical examination of the blood would possibly be diagnosed as quotidian) is somewhat striking.

MIXED INFECTION.

Several cases of mixed infection, the parasites being those of tertian and of æstivo-autumnal fever, were met with, but their order with reference to date of infection could not be made out.

No cases of hæmoglobinuric fever have ever come under my notice during a seven years' residence in the island, and I have had no case under quinine treatment where this drug caused hæmaturia.

TREATMENT.

The interest attaching to the recent discussion in the *British Medical Journal* with reference to the

hypodermic injection of quinine as a treatment of malaria makes me digress from the subject proper of this article to say a word in this connection. While quinine in solution, and given by the mouth, has been my usual method of treatment, I have, in cases specially resistant to quinine in this form, or where head symptoms were prominent, used the acid hydrochloride of quinine hypodermically, or rather intramuscularly—the smallest dose given for an adult has been 3 gr. night and morning for three days, six doses in all; the largest single dose given in any case was 10 gr.; the usual dose given was 5 gr. night and morning for three days; the results have been most satisfactory. My experience has been that quinine given in this form does not upset digestion nor cause cinchonism, as when given by the mouth, while its specific action is more readily and completely obtained.

TABLE III.—SHOWING TERTIAN AND QUARTAN MULTIPLE INFECTIONS.

| | TERTIAN | | QUARTAN | | |
|--------------|--------------------|----------------|--------------------|----------------|----------------|
| | Total No. of Cases | Double Tertian | Total No. of Cases | Double Quartan | Triple Quartan |
| 1901. | | | | | |
| February .. | 4 | — | 3 | — | 1 |
| March .. | 3 | — | 4 | — | 2 |
| April .. | 4 | 2 | 1 | — | 1 |
| May .. | 2 | — | 1 | 1 | — |
| June .. | 2 | 2 | 1 | — | 1 |
| July .. | 68 | 48 | 2 | — | 1 |
| August .. | 38 | 20 | 3 | — | 1 |
| September .. | 34 | 21 | 3 | 2 | — |
| October .. | 22 | 8 | 4 | — | — |
| November .. | 7 | 2 | 2 | — | — |
| December .. | 12 | 4 | 6 | 4 | 1 |
| 1902. | | | | | |
| January .. | 9 | — | 4 | — | — |
| Total .. | 205 | 107 | 34 | 7 | 8 |

ANOPHELES.

Anopheles occur and have been found at many places in the island, and are most plentiful in the most malarious parts. Some specimens were sent to Dr. Daniels, who very kindly undertook to have them identified. They were found to be *Anopheles maculipennis*, and this is certainly the commonest species here. I have also seen a smaller and darker coloured *Anopheles*, but do not know to which species it belongs.

I trust that these statistics, meagre as they are, and covering as they do only a short period, may be of some use and interest with reference to malaria in Cyprus.

REFERENCES.

- ¹ Malaria according to the New Researches. By Professor Angelo Celli. Translated by J. J. Eyre, M.R.C.P., p. 157.
- ² Hygiene and Diseases of Warm Climates. By Andrew Davidson, M.D., p. 121. ³ *Op. cit.*, p. 149. ⁴ Report of the Malaria Expedition to West Africa, August, 1899. Liverpool School of Tropical Medicine, p. 6. ⁵ Davidson, *op. cit.*, p. 139. ⁶ Davidson, *op. cit.*, p. 137. ⁷ *British Medical Journal*, May 3rd, 1902, p. 1113.

EGYPTIAN MEDICAL CONGRESS.

THE following gentlemen have kindly consented to join the Committee in England on behalf of the Egyptian Medical Congress, which will be held at Cairo on December 19th-23rd next. Special facilities and reduction in fares are arranged for members wishing to be present at the Congress. Anyone wishing for further information will please apply to any member of the Committee, or to the Hon. Secretary, 9, Manchester Square, London.

List of the Committee: Sir Lauder Brunton, Mr. Watson Cheyne, Sir William Church, Bart., Mr. Reginald Harrison, Sir Victor Horsley, Dr. Stephen Mackenzie, Dr. Page May (Hon. Sec.), Sir Douglas Powell, Bart., Dr. George Savage, Sir Thomas Smith, Bart., Dr. James Taylor, and Sir Frederick Treves, Bart.

The British Guiana Medical Annual for 1902.

ANKYLOSTOMIASIS; IS PIGMENTATION OF THE TONGUE AN EARLY SIGN?

By P. H. DELAMERE, L.R.C.P.I.

THE early diagnosis of this grave and fatal disease being of such vital importance to both the employer of East Indian labour and the medical officer in charge, any new or hitherto unrecognised sign of early disease will, I feel certain, be of special interest. This is therefore my reason for bringing before the medical profession a symptom which I believe to be an early sign of ankylostomiasis.

Every writer on ankylostomiasis lays stress on the importance of early diagnosis, to quote Manson's "Tropical Diseases" only, he says, "*Provided its presence be suspected ankylostomiasis is easily diagnosed.*"

"In tropical countries anæmia without apparent cause should always suggest a microscopical examination of the fæces." But I think I have found an earlier sign, viz., before that of anæmia, and it is with the hope of producing investigation, and proving or disproving the theory, that I place before the readers of this journal this short paper. If I am correct, then it is a very easy diagnosis and one that can be made at once and anywhere.

Every day that an immigrant is in hospital there is a distinct loss both to the estate that employs him and to the immigrant himself, and in these hard times it becomes a burden, when say on any one estate ten to twenty labourers are in hospital for weeks or even months at a time from this disease. The loss becomes heavy, the man loses a shilling a day, and the estate another shilling or more; and when this is multiplied by the daily loss all over the Colony the figures made up will be found to be very great.

It therefore becomes a most urgent question to save some of this loss at any rate, and as every one will agree that: (1) ankylostomiasis is the cause of a large number of detentions in hospital; (2) in its early stages it is certainly curable. It is idle to say therefore that an early sign of this grave disease would not be of the utmost value to every one concerned.

Some few months back I one day in examining

a patient noted that his tongue was marked all down one side just as if he had wiped a penful of Stephen's blue-black ink on it; I chided this man for playing with the pens and ink that were on the ward table. He assured me he had never touched them, and on further examination I found that they were not ink stains. He said his tongue had been marked like that for a long time; now this man was not anæmic even to a slight degree, came in for slight fever, his organs were healthy, there was no outward sign of ankylostomiasis. Now in cases like this, one does not (or shall I say did not then), examine the fæces for more than one reason easily understood. This man got over his fever and was discharged. Shortly after another case came in, anæmic and with large blue-black marks on the tongue; his general appearance gave me the idea that he had ankylostomiasis. Not having a microscope I treated him on chance with thymol in the usual way, as suggested in a recent circular on the subject from the Surgeon-General, and on having the stools examined after thymol several ankylostomes were found and shown to me. I then connected the two cases, got my former patient back, and he, also, after treatment with thymol passed ankylostomes. After that I looked at every tongue I came across, and at one monthly inspection found no less than fourteen marked tongues, some with only two or three very small blue-black marks less than a sixteenth of an inch, and of irregular shape, some like little islands of blue-black in the centre of a perfectly healthy-looking tongue, others with a strip down one side of blue-black, and all in perfectly healthy non-malarial first year's coolies. They were all treated with thymol and every one passed matured ankylostomes, and on further treatment left the hospital with the marks removed (I find that the marks after thymol treatment begin to fade at the edge and gradually lose colour, getting paler till they fade away). Later on I found three cases that had brown marks on the tongue, in two minute coffee-coloured spots at tip, and the other a large coffee-coloured mark on the side of the tongue with minute dark brown spots in it. These also after thymol passed ankylostomes, but the patients were anæmic when first seen.

Now if these cases mentioned had been allowed to go on till symptoms of anæmia showed, the patients would have been in a more advanced stage of the disease; as I hold that they were suffering from ankylostomiasis, they all having passed ankylostomes, and consequently taken twice or three times as long curing, and twice or three times as great a loss to the estate, or been past curing and become candidates for that gang of much to be regretted "invalids" that one finds on every estate, useless and expensive to the estate, and a continued source of trouble to the Medical Officer and Immigration Agent.

I have been told on making known this theory that the marks are caused by malaria, and not by the ankylostome. Of course malaria is much more common than ankylostomiasis. There were 843 cases of malaria treated in the estate hospital under my charge during 1901-2, besides a greater number as out-patients. Now if malaria causes this marking, one should find many more cases of it than I have come across. Thirty-eight were treated in hospital,

and several seen in private practice, and is it not strange that if this is not a sign of ankylostomiasis, each one of my freshly-imported, healthy-looking, and certainly non-malarial cases, should have passed ankylostomes after thymol. That malaria does mark the tongue I know, but I have only found it in advanced cases of malarial cachexia, not in strong, healthy adults with no sign of disease about them; if it does, then I should have found hundreds of cases by this time.

Daniels found on microscopic examination that there was a distinct pigmentation of liver, kidney, and sometimes spleen caused by the ankylostomes (*B. G. Annual*, 1895, p. 21) which was not malarial. Now if malaria causes a pigmentation of spleen, &c., as well as of the tongue, why should not the toxic substance which he believes is produced by the ankylostome, and absorbed from the bowel, causing blood destruction, produce pigmentation of the tongue, as well as of liver, &c.

In another way it becomes an important sign; the early symptoms of this disease, before anæmia shows itself, as laid down in the works on the subject, are dyspeptic troubles and pain or uneasiness in the epigastrium. Now with a class of patients such as one usually sees in an Estate hospital, it is anything but an easy matter to get an intelligent history of symptoms; there is first the language difficulty, and if the patient speaks any English "a pain in me bellie" may mean anything, and "a cross pain," &c., will not help, but until the anæmia puts you on the track, there is little to point to the presence of ankylostomes, unless everyone's stools are examined, which though giving big results takes time, but with a blue-black or brown mark on the tongue one is on the track right away, and can hardly help being aware of the patient's state, without even asking for symptoms. The patients do not look or feel ill, but there is the sign of this grave disease lurking in their system, which if not treated will shortly make them useless and expensive members of the community.

That the disease is on the increase in this island I am quite certain, as not only have I seen it in the Estate hospitals, but I have seen many cases in my private practice.

Current Literature.

THE TREATMENT OF PERNICIOUS FORMS OF MALARIAL FEVER.—Professor Montoro de Francesco, as the result of observations of certain severe forms of malaria in Calabria, Italy, which occur chiefly in summer and in autumn, advocates hypodermic injections of quinine. He remarks that the temperature will often rise after repeated small doses of quinine. In severe types of malaria, Professor Francesco advises that 30 grains of the dihydrochlorate of quinine should be administered hypodermically to begin with, and then 16 grains daily until the patient has been free from fever for three or four days. Afterwards 16 grains of euquinine should be given internally every day for one month. Francesco advocates and prefers euquinine owing to its tastelessness, and that no unpleasant symptoms result

from its use. In the case of children euquinine is especially valuable.—*Klin-therap. Woch.*, 1902, Nos. 23-25.

ANKYLOSTOMIASIS IN AN INDIVIDUAL PRESENTING ALL OF THE TYPICAL SYMPTOMS OF PELLAGRA.—H. F. Harris reports this interesting case. The patient, a man, aged 29, lives in Georgia. He has been brought up in great poverty. His bread has always been prepared from Indian corn, which has formed the greater part of his diet. The first symptoms usually begin in the spring with loss of appetite, thirst, and a feeling of malaise. He suffers from melancholia. His legs become extremely weak, and sensation to pain is far below normal. In the spring his hands, arms, and the dorsal surfaces of his feet become greatly inflamed. Blisters form, followed by scabs. He suffers from constipation, and has a great distaste for food, especially for Indian-corn bread. During these periods he has constant pain in the neck and tenderness and pain in the gastric region. He vomits frequently. He begins to improve after May or June, and by July or August is generally well again. The patient has all of the typical symptoms of pellagra. If this is a genuine example of the disease, it is the first case of the kind that has been reported in the United States. In the man's faeces, numerous eggs of the ankylostoma have been found, and it seems not unlikely that the pathologic condition is in large measure due to this cause; but, on the other hand, there are certain features that are not consistent with this explanation, such as the strange tendency of the disease to manifest itself only in the spring and early summer, and the fact that it has existed for fourteen years. These parasites are said not to live in the body for more than five or eight years at the very outside, and unless the patient has been repeatedly re-infected, the presence of these parasites cannot entirely explain the long duration of the disease. Thymol was recently given to the patient, and not less than 600 worms passed from the bowels. No eggs of the ankylostoma were found four weeks after the treatment. The patient, however, is now feeling no better than before. If he had pellagra, the disease is so far advanced that nothing probably could be done for him. He has been advised to go to a cooler climate, and to be careful not to eat decomposed Indian corn. It should be noted that the blood examination showed the hæmoglobin to be only 20 per cent., the red cells 1,760,000, and the white cells 4,020; there was decided poikilocytosis.—*American Medicine*, June 19th, 1902.

PLAGUE.

INDIA.—During the weeks ending September 27th and October 4th, the deaths from plague in India number 9,976 and 10,718 respectively. The mortality during the latter week occurred as follows: Bombay City, 101; Presidency, 7,780; Karachi, 23; Madras, 385; Calcutta, 9; Bengal, 52; United Provinces, 736; Punjab, 210; Central Provinces, 2; Mysore, 1,122; Hyderabad, 149; Berar, 148; and Central India, 1.

EGYPT.—During the week ending October 12th, one fresh case of plague was reported in Alexandria. Since October 10th Egypt has been entirely free of plague.

MAURITIUS.—During the week ending October 23rd, 17 fresh cases of plague and 11 deaths from the disease were recorded in Mauritius. During the week ending October 30th, there occurred 29 cases of plague and 16 deaths from the disease.

HONG KONG.—One case of plague only was notified in Hong Kong during the week ending October 25th.

RUSSIA.—Since the recurrence of plague in Odessa, in May, 1902, 15 deaths from plague only have been recorded. The Russian authorities are taking extraordinary precautions against the spread of the disease.

PALESTINE.—During the week ending November 1st, 494 deaths from plague occurred at Gaza and the neighbouring villages, 78 at Lydda, and 5 at Jaffa.

CHOLERA.

EGYPT.—During the week ending October 13th, 819 cases of cholera occurred in Egypt against 1,571 during the previous week. During the week ending October 20th, 685 cases were reported.

SYRIA.—For ten days previous to October 18th, 40 deaths from cholera were stated to have occurred in Syria.

LEPROSY: POSSIBLE SOURCES OF CONTAGION.—Gravaqua has found the bacillus of leprosy on the surface of leprosy lesions, both recent and healed, and on gold coins which have been handled by lepers. —*Journ. de Mal. Art. et Syph.*, January, 1902.

BLACKWATER FEVER IN THE PHILIPPINES.—In the *New York Medical Journal* of September 13th, 1902, Dr. F. M. Hartsock describes the clinical symptoms and the *post-mortem* signs of what appears to be a typical case of blackwater fever occurring in Manila.

BIRTH.

ON September 19th, the wife of Professor W. J. Simpson, M.D., F.R.C.P., of a daughter.

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Médecine Navale.
Archives Russes de Pathologie, de Médec. Clinique et de Bacteriologie.
Australasian Medical Gazette.
Boletín de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Brooklyn Medical Journal.
Caducée.
Climate.

Clinical Journal.
Clinical Review.
Giornale Medico del R. Esercito.
Hong Kong Telegraph.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
Journal of the American Medical Association.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Medical Record.
Medical Review.
Merck's Archives.
New York Medical Journal.
New York Post-Graduate.
Pacific Medical Journal.
Polyclinic.
Public Health.
Revista de Medicina Tropical.
Revista Médica de S. Paulo.
Sei-i-Kwai Medical Journal.
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The Journal of Tropical Medicine.

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Original Communications.

NOTES OF A TOUR IN THE NORTH CANARA DISTRICT OF INDIA IN SEARCH OF MOSQUITOES.

By E. H. AITKEN.

(Continued from p. 327, November 1st, 1902.)

It remains to give some account of the mosquitoes collected during the tour. I have already referred to the difficulties arising from limited time, rapid travelling, and the want of conveniences for rearing and watching larvæ of many different species at one time. These hampered me so much that I was able to rear only a very few specimens of each species and lost some altogether, so the collection I brought back with me numbered only forty-eight specimens belonging to thirteen or fourteen species. I had preserved the larvæ of five of these and of course kept notes, the substance of which I give below.

To begin with *Anopheles*, to which the other genera owe all the interest that they have recently excited, I obtained six species, three of which I identified as *A. rossii*, *A. barbirostris* and *A. christophersi*. The fourth is a species allied to *A. Jamesii* (Theobald). It is probably the African species *A. maculipalpis* (Giles). The fifth is, I think, a new species allied to *A. maculata* (Theobald). The sixth was a minute species with unspotted wings.¹ All these were bred, and of course it was my first object to learn as much as I could about the life of each in its earlier stages. To this end the first step was to learn to distinguish the larvæ at sight if possible. The best method to attain this skill is to put your larvæ into shallow vessels (white saucers do well), and stare hard at them by the hour until you come to recognise them, as you recognise a friend when he is too far off for his features to be distinguishable. I was soon able to divide my larvæ into three groups, readily distinguishable from each

other, which I will call the *rossii*, *christophersi* and *barbirostris* groups. *A. rossii* is the commonest species in Bombay, and its larva was already very familiar to me, so I will take it as the standard of comparison. It is not only the commonest but also the easiest to find, always on the surface, darting about backwards, and snapping at its neighbours. It does dive, of course, when threatened, but comes up again very soon. When young it is brown, with a blackish head and a white collar. When full grown the colour is variable, light brown, almost black, or greenish, the head being generally mottled brown. Just before becoming a pupa it turns pale and looks very fat and smooth; at this stage I have taken it for a different species. The larva of *A. barbirostris* is markedly different. It is usually very dark in colour, with a light collar and a light band at the third abdominal segment, most prominent in the young. Sometimes there is a broad silvery, dorsal stripe; but colour is a very unsafe guide. Form and habitual attitude can be trusted, and, in the case of *barbirostris*, serve to distinguish it almost at a glance from all larvæ of the *rossii* type. The thorax is small, scarcely exceeding the head in breadth, the head is elongated, and the abdominal segments are nearly equal, so that the insect looks long and worm-like when compared with the larvæ of *rossii*. This appearance is enhanced by its attitudes, which are less rigid, even when it is floating at rest. When browsing on *confervæ*, which appear to be its principal food, it lies like a snake. It appears to feed very little on the surface. I found this species in a rocky pool in one stream and among dense grass and weeds in another.

The larva of *A. christophersi* differs as markedly from that of *rossii*, but the differences take just the opposite direction. The head and thorax are broad, the abdominal segments decrease rapidly from the first to the last and are very sharply defined. The lateral bristles are long and stout. But the most definite mark is the shape of the thorax, which is not round or oval, but distinctly quadrilateral and broader behind than before. The back of the head is as broad as the front of the thorax, so that the two together

¹ This is probably the new species I am describing as *A. im-maculata*. F. V. Theobald.

form a rough triangle. With a little practice this becomes very readily recognisable, whether the larva is old or young. The colour is usually light greenish-brown, the head being darker. These larvæ are very alert, diving on the least alarm and lying motionless at the bottom. For catching them I found nothing equal to a white teacup of enamelled iron. If dipped adroitly it engulfs them before they have time to escape. But if any mud has been taken in with the water, it is most difficult to detect them, so patiently do they sham death. They do not seem to feed on *confervæ* at all. If anything of the kind is put into the water with them, they are apt to entangle themselves and die, by drowning I suppose. They feed either on the surface or at the bottom, twirling their brushes. For this reason, perhaps, they affect shallow, clean water. I found them in rice-fields and small rocky streams, but most abundantly in boggy ground adjoining rice-fields. The larva of the *Anopheles* with unspotted wings belongs to this type. I could distinguish the two when I saw them together, but cannot describe the difference. I obtained very few of the unspotted species, all in one pool formed by a stream in a dark forest.

The differences which I have attempted to describe in the three types of larvæ seem to correspond to differences just as marked in the imagines. The *Rossii* group are typical *Anopheles*,¹ *Barbirostris* is unique, while the *Christophersi* group comprises small forms more resembling *Culex* in their outline and habitual attitude.

Of *Culices* other than *Anopheles* I got some interesting species. The commonest larva above the Ghauts was that of *Culex fatigans*, which I found keeping company with *Anopheles* everywhere. I need not describe it. Many contradictory statements have been made about the larvæ of *Culex* destroying those of *Anopheles*, and *vice versa*, based probably on observations made upon very different insects which have been lumped together as *Culex*, so I will give here the results of my own experience. I believe that the larva of *C. fatigans* and every other of the same type is purely vegetarian. The larvæ of *A. rossii* feed greedily on the dead bodies of their own species, and I am almost certain that they will kill and devour weak or sickly individuals; but the larvæ of *C. fatigans* live as amicably together as a flock of sheep. There is, however, another mosquito, strangely like *C. fatigans* at first sight, but twice as large, the larva of which is very different both in aspect and temper. It is a creature of rather striking appearance. The head is brown, but the body is usually of a transparent white colour, except the posterior half of the thorax, the third, sixth and last segments of the abdomen and a central line, which are of a rich brown hue. It floats horizontally, but of course not at the surface, the breathing tube being moderately long. This creature feeds, as far as I have seen, exclusively on other larvæ, catching them by any part and chewing them up, undisturbed by their wriggings. I fed mine on *Culex* and *Anopheles*, but they devoured each other also till there were only two monsters of equal size left, which produced fine female mosquitoes. I got more afterwards. I found this species, with other

larvæ, in grassy pools, and once in a very deep well. I have also taken them from a well in Bombay. As might be inferred from its habits, this larva differs in many points from that of a typical *Culex*. The most obvious difference, and one that can be seen with the naked eye, is in the antennæ. Those of *C. fatigans* are long and tufted at the ends and, standing out like the jaws of a stag beetle, give the insect a fierce aspect. In the larvæ I am describing they are as short as in *Anopheles* and must be looked for with a lens. The jaws are formidable, and the brushes, which are large and consist of very stout bristles combed at the inner edge, do not overhang the mouth, but extend laterally. I never saw them in motion and their position suggests that their function may rather be to assist in holding prey. I have said that the mosquito is very like *C. fatigans* in form and colour, but the femora and tibiæ are speckled with yellow. I have no means of identifying it at present and must leave its name to a foot-note.¹

Another rather striking larva, with banded body and very large head, which I found commonly in grassy pools along with those of *Anopheles* and *Culex*, surprised me by producing an *Aedes*. With its long breathing tube and large, tufted antennæ, it closely resembled *C. fatigans*, except in colour, but it floated horizontally, though, of course, at some distance below the surface. It appeared to feed entirely at the surface, after the manner of *Anopheles*. I regret that I did not examine the pupa carefully. The imago, when it came, was a minute dusky gnat, which I took for the unspotted *Anopheles* mentioned above until I had examined the palpi. I tried to get it to taste my blood, but in vain. Its name must be relegated to a foot-note also.²

I reared a good many *Stegomyia* larvæ. I caught *S. scutellaris* biting one of my peons in a field in broad daylight, but did not find its larvæ. I have reared it in Bombay, however, and may say that there is little difference between the two larvæ in form or habits, in both of which they differ from *Culex* proper. The antennæ are short and straight, the head not so broad as the thorax, and the breathing tube short and stout. Their position when floating is nearly perpendicular. Their food consists largely of rotting leaves, and the rapidity with which some in my keeping reduced a leaf to a skeleton suggested that this might be one of the ways in which the beautiful skeleton leaves, which one often meets with in this country, are produced. Colonel Giles speaks of *Stegomyia* as essentially a monsoon mosquito, and so it is on the plains, where the conditions it requires are not obtainable at any other season. The ideal breeding place for this genus is a small hole in a rock, or in a hollow tree, well shaded from the sun and filled with a brew of rotting leaves the colour of beer. In Canara this can be had at any season, and I found the gloomy beds of forest streams swarming with them in March and April. They were venomous and thirsty, and having

¹ This mosquito is *Culex concolor* (Desvoidy). The characteristic position assumed by the larvæ in water and their cannibal habits have been previously noted (cf. "A Monograph of the Culicidæ," by F. V. Theobald, vol. ii. p. 110).—S. P. James.

² I have not seen this species; it is probably a new one.—F. V. T.

¹ *Rossii* is not a typical *Anopheles*.—F. V. T.

once found me they would follow me out into the sunlight and refuse to be driven off.

I found some other curious larvæ which I did not succeed in rearing, and some mosquitoes of which I failed to get larvæ. A fine female of *C. mimeticus* emerged before my eyes from a pupa in a morning's "bag" which I had not yet sorted. I got only one other specimen and that I caught in a hut. I never saw a *Megarrhina*. I had not much leisure to look for them, and possibly they were not on the wing at that season. I was also singularly unsuccessful in getting eggs, but I did not regret this much, because in the circumstances I could not possibly have reared mosquitoes from them (a difficult thing at the best), and without doing this I could not have been sure of the species to which they belonged.

I will not apologise for giving no detailed and minute descriptions. I have put my collection into hands much abler for that kind of work than mine. These are field-notes merely. I will conclude by mentioning one fact which it may be worth the while of collectors to note. A mosquito does not attain its proper colour for some time after emerging from the pupa, and collectors may prepare much perplexity for themselves by being in too great haste to kill their specimens. Of two specimens otherwise alike one may have the femora and tibiæ almost white, not because it is a different species, but because it was killed sooner.

SLEEPING SICKNESS: A SUGGESTION.

By ALEXANDER CROMBIE, M.D.Edin., C.B.

Lieut.-Col. I.M.S. (retired).

THE exceeding prevalence of *Filaria perstans* in the blood of the inhabitants of districts where sleeping sickness is endemic, the presence of the parasite in the only three cases he had seen in England, the correspondence which obtains between the geographical distribution of the disease and that of the parasite, and the fact that the parasite may remain alive and the disease manifest itself years after the endemic area has been quitted, led Manson to suggest that the parasite was in some way responsible for the disease; though it seemed to him hard to say in what way it operates. ("Tropical Diseases," 1900, p. 286.)

It has been objected to this theory that the parasite may exist in the blood in enormous numbers without any symptoms of this or any other disease, and on the other hand, that sleeping sickness may exist and proceed to a fatal termination with few or no parasites in the blood, and that indeed towards the end of an attack they are generally absent.

The only legitimate deduction which could be made from such data is that African lethargy is not due to a toxin elaborated by the parasite circulating in the blood like an opiate, and dependent, as regards its intensity, on and commensurate with, the degree of filarial activity present.

These cannot indeed be regarded as serious objections, because the forms of the parasite found in the blood are the embryos, and taking *Filaria nocturna* as an analogue, we may assert that the embryos are

probably not of any pathological significance. The same objections could be made to considering chyluria, or elephantiasis, to be filarial diseases; they bear no relation to the number of embryos in the blood, and indeed in elephantiasis it is exceptional to find any. These diseases are conditional on the position in the lymphatic system taken up by the parent worm and the consequent obstruction to the lymphatic flow. The parent worm, which was the original cause of the obstruction, may die, and the embryos disappear from the blood, but the effects of its operation may be permanent.

So it may be with sleeping sickness. The disease, if on all fours with chyluria and elephantiasis, would not bear any relationship to the number of embryos found in the blood, but would be determined by the position occupied by the parent worm and the mechanical difficulties which it might cause in that position.

Hitherto, the parental forms of *Filaria perstans* have been found only in the connective tissue at the root of the mesentery, behind the abdominal aorta, and beneath the pericardium (Manson). It is obvious that they are incapable in these positions of giving rise mechanically to the symptoms of sleeping sickness. To do this they must be sought for elsewhere.

It would be vain to look for them in the encephalon itself. No gross lesions such as would be necessary to cause the symptoms have been found within the cranium, and moreover, localised lesions such as they would produce would be betrayed by localised and partial symptoms, which are generally absent in sleeping sickness. It may be ushered in by epileptiform seizures or maniacal attacks, but more usually it begins with listlessness, headache, giddiness, lassitude, and a tendency to somnolence, which gradually deepens; symptoms of a cause affecting the brain generally and not locally.

Mott has shown (*British Medical Journal*, Dec. 16th, 1899) that naked-eye abnormalities in the brain are almost nil, but microscopic sections show that the essential condition is crowding of the pia mater and perivascular spaces with mononuclear leucocytes. The perivascular spaces constitute the lymphatic system of the brain, and we must therefore conclude that as in chyluria, elephantiasis, &c., the essential condition in sleeping sickness is one affecting the lymphatic system.

The most constant symptom in sleeping sickness, as well as in infection by *Filaria perstans*, with or without somnolence, is enlargement of the lymphatics, in sleeping sickness those of the neck being chiefly affected. It is said to occur in 99 per cent. of the cases. The enlargement and obstruction of the lymphatic glands of the neck would account for the fullness and puffiness of the face, which is noticed early in the disease, and also possibly for the tremors of the lips and tongue, the lymphatic vessels from these parts all ending in the superficial lymphatics of the neck, and it is probable that the parent form of the worm, or its aborted ova, will, if sought for there, be found in or around these vessels, or glands, giving rise, as in the case of *Filaria nocturna*, to lesions analogous to those which cause chyluria, elephantiasis, and lymph-scrotum.

But enlargement and obstruction of the superficial lymphatic glands of the neck would not affect the lymphatic flow within the cranium, and could not cause the brain symptoms which characterise sleeping sickness. The lymphatics of the brain leave the cranium through the jugular foramen, with the jugular vein, and join the upper set of the deep cervical glands which extend along the course of the internal jugular vein from the base of the skull to the level of the thyroid cartilage. Obstruction of this set of glands would cause lymph-stasis within the cranium, changes in the perivascular spaces, and probably all the phenomena of sleeping sickness. The periods of temporary abatement of the symptoms which often mark the earlier stages of the disease would correspond with periods of lessened inflammatory obstruction.

I would suggest, therefore, to those engaged in the investigation of this disease, that in every fatal case of sleeping sickness, the deep cervical glands, both the upper and lower sets, of both sides, should be carefully examined for signs of disease, as being the most likely seat, on *a priori* grounds, of the essential condition, probably caused by the presence of the parental form, or its aborted ova, which determines the occurrence of sleeping sickness in cases of infection by *Filaria perstans*.

I venture to think that the deep cervical lymphatics are rarely examined in *post-mortem* researches, but the reasoning by exclusion which I have adopted seems to narrow down the cause of the symptoms to disease affecting the glands which receive the lymph from the brain, as the only ones whose obstruction would presumably give rise to the phenomena of sleeping sickness. I offer the suggestion for what it is worth as a working hypothesis of sufficient reasonableness to draw attention to the condition of these glands in future investigations into a disease which has hitherto eluded elucidation. I have ignored in this paper the other theories which have been advanced to account for it, as they fail to satisfy completely the conditions which Manson has pointed out as indicating a connection between sleeping sickness and the prevalence of *Filaria perstans*, and my only object is to draw attention to a hypothesis which would be in complete accord with all we know of the disease, if the deep cervical glands were found in all fatal cases to be the seat of inflammatory and obstructive changes caused by the presence, or former presence, of the parent worm. I must leave the suggestion I make in the hands of those who have opportunities, which I do not possess, of investigating this particular point, and only venture to think that if the parent worm, or the results of its presence, are not found in the place indicated, it will be time enough to cast about for some other explanation.

admitted to hospital, where he stated that the pain in the foot was very bad, and gradually decreased up to the knee, where it was slight. The pain subsequently extended to the right groin, and the superficial and deep reflexes of the right leg were found to be slightly exaggerated. As the patient was getting worse, Lieutenant Murison injected 5 cc. of Calmette's antivenene, the bite being cauterised with silver nitrate, and a 1 in 40 carbolic dressing applied. In fifteen minutes afterwards the pain began to decrease, but the patient being very drowsy the attendants had some difficulty in keeping him awake. In five days from admission the patient was discharged from hospital cured.—*Indian Med. Gazette*, May, 1902.

YELLOW FEVER AND MOSQUITOES.—In an interesting paper by Edmund Souchon, M.D., in the *Medical Record* of October 25th, 1902, on the subject of "The Eradication of Yellow Fever in Havana," the author gives a list of the epidemics of yellow fever in New Orleans from 1817 to 1899 inclusive—eighty-three years:—

| YEAR. | DEATHS. | YEAR. | DEATHS. | YEAR. | DEATHS. |
|------------|---------|------------|---------|------------|---------|
| 1817 | 809 | 1845 | 2 | 1873 | 226 |
| 1818 | 115 | 1846 | 160 | 1874 | 11 |
| 1819 | 2,190 | 1847 | 2,359 | 1875 | 61 |
| 1820 | 400 | 1848 | 808 | 1876 | 42 |
| 1821 | — | 1849 | 769 | 1877 | 1 |
| 1822 | 239 | 1850 | 109 | 1878 | 4,046 |
| 1823 | 1 | 1851 | 17 | 1879 | 19 |
| 1824 | 108 | 1852 | 456 | 1880 | 2 |
| 1825 | 49 | 1853 | 7,970 | 1881 | 0 |
| 1826 | 5 | 1854 | 2,423 | 1882 | 1 |
| 1827 | 109 | 1855 | 2,670 | 1883 | 4 |
| 1828 | 130 | 1856 | 74 | 1884 | 1 |
| 1829 | 215 | 1857 | 199 | 1885 | — |
| 1830 | 117 | 1858 | 4,855 | 1886 | — |
| 1831 | 2 | 1859 | 91 | 1887 | — |
| 1832 | 18 | 1860 | 15 | 1888 | — |
| 1833 | 210 | 1861 | — | 1889 | — |
| 1834 | 95 | 1862 | 2 | 1890 | — |
| 1835 | 284 | 1863 | 2 | 1891 | — |
| 1836 | 5 | 1864 | 6 | 1892 | — |
| 1837 | 442 | 1865 | 1 | 1893 | — |
| 1838 | 17 | 1866 | 192 | 1894 | — |
| 1839 | 452 | 1867 | 3,107 | 1895 | — |
| 1840 | 3 | 1868 | 5 | 1896 | — |
| 1841 | 594 | 1869 | 3 | 1897 | 297 |
| 1842 | 211 | 1870 | 588 | 1898 | 57 |
| 1843 | 487 | 1871 | 55 | 1899 | 23 |
| 1844 | 148 | 1872 | 40 | | |

He strenuously opposes Dr. Gorga's contentions that reliance should be placed on the destruction of the mosquitoes, which would practically mean the abolition of quarantine. Though the author has a thorough belief in the transmission of yellow fever by mosquitoes, as demonstrated by the memorable labours of Dr. Walter Reed, his co-workers and followers, he believes, from practical facts before him, that there is some other means of transmission.

Dr. Souchon concludes his article: "Until these facts are satisfactorily explained and demonstrated to us, we, of the Louisiana State Board of Health, the guardians of the health of the city of New Orleans, the State of Louisiana and of the whole Mississippi Valley, cannot think of altering the present maritime quarantine regulations."

SNAKE-BITE ; TREATMENT OF.—Lieutenant C. C. Murison, I.M.S., reports a case of snake-bite (*Echis Carinata* [Fursa]) in a boy, aged 12. About half an hour after the bite the site of the injury—the back of the foot—became painful and swelled up. He was

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THE

Journal of Tropical Medicine

NOVEMBER 15, 1902.

RESEARCH LABORATORIES IN KHARTOUM.

THE announcement that research laboratories are to be founded at Gordon's Memorial College, Khartoum, is an important step in the progress of the civilisation of the Soudan. The chemical and bacteriological laboratories about to be opened there are the gift of Henry S. Wellcome, Esq., of the well-known firm of Messrs. Burroughs Wellcome and Co., of London. The environment of Khartoum is an unknown factor so far as disease is concerned, and important results are likely to reward the munificent founder of the Institution.

Standing as it does as a watch tower between Central Africa and the Nile Valley, Khartoum will become in the near future, a political and economic factor of far-reaching consequence ; and the influence of Gordon's Memorial College, inasmuch as it promises to be a centre of scien-

tific education and research, will add immensely to the prestige of this singularly situated city.

The Colonial and Foreign Offices, under their present directorates, have come to grasp the importance of the health of the people, both native and foreign, as an economic factor in the development of every tropical and sub-tropical country ; and the soundness of this doctrine is already bearing fruit in many of our colonies and protectorates.

We are specially interested at the present moment in the advance and spread of several ailments, and in none more so than in that of sleeping sickness. We have from time to time drawn attention to the fact that this disease has crossed the high plateau between the valley and the Congo and the upper reaches of the Nile, and seems to be devastating countries in which, until the last few months, the disease had been unknown. Should the spread continue, the inhabitants of the Nile Valley and all between Uganda and the coast are threatened with this deadly scourge, and it is of the utmost consequence that a watch should be kept upon its advance. For this reason alone we welcome the establishment of scientific laboratories in the Eastern Soudan ; and this is but one example, of many that might be adduced, to prove the vital necessity of the application of modern research laboratories as guardians of the public health in outlying regions of civilisation.

We hope a brilliant future awaits this recently established seat of science, and when we consider the obscurity that prevails concerning the Dark Continent, be it of its peoples, its agriculture, or its diseases, we are sure that the hope we entertain, that the obscurity will speedily be dispelled, is well founded. It is gratifying to know that so able a Director has been chosen to superintend the research laboratories. Dr. Andrew Balfour is so well known as a reliable and careful investigator, that the work is sure to be well done ; and as the material at his command is illimitable, we are certain to have additions to our scientific knowledge of lasting benefit and importance. The natives of the Soudan owe a lasting debt of gratitude to Mr. Wellcome for

his public-spirited and philanthropic gift; and all students of tropical medicine are indebted to him for the opportunity given them of elucidating many of the pathological problems which, owing to imperfect means of obtaining information, are still unsolved.

British Medical Association.

MEDITERRANEAN FEVER.

By Staff-Surgeon P. W. BASSETT-SMITH, R.N.
Lecturer on Tropical Diseases, Haslar.

THE AGGLUTINATING PROPERTIES IN THE BLOOD IN CASES OF MEDITERRANEAN FEVER, WITH SPECIAL REGARD TO PROGNOSIS, AND REMARKS ON OTHER BLOOD CHANGES AND REACTIONS DURING THE COURSE OF THE DISEASE.

WHILE in charge of wards for the treatment of tropical diseases at the Royal Naval Hospital, Haslar, the number of cases admitted suffering from Mediterranean fever during the past two years and a half has been considerable (196). This disease, which causes such a drain on the naval forces, particularly on the Mediterranean station, has from its importance demanded particular attention. The cases received are mostly in the persons of officers and men who have been under treatment for a more or less prolonged

tions which are here recorded. It was in 1887 that Bruce isolated and definitely ascertained that the specific cause of the disease was due to the presence of a minute micrococcus abundantly present in the spleen both in the living and dead. Since that time his observations have been repeatedly confirmed. In 1901 I obtained a pure culture of the organism from the spleen removed three hours after death of a fatal case which occurred here, and from which and other strains the agglutination reactions have been made. Bruce at Malta, Wright and Semple at Netley, found that the organism injected into monkeys produced a fever like that of the human subject, and from them again isolated the same organism. Unfortunately the specific action of the micrococcus *melitensis* has been too frequently demonstrated by inoculation, accidental and otherwise, in laboratory work, the following cases being recorded:—

| | | | | |
|----------------|--------------|---------------|---------------|---------|
| 1897..A.E.W. | .. Netley .. | Purposeful .. | Incubation .. | 15 days |
| | | inoculation | | |
| 1897..S. | | Accidental .. | | 15 .. |
| | | inoculation | | |
| 1899..Corp. S. | | " .. | " .. | — |
| 1900..B. S. | .. Haslar .. | " .. | " .. | 6 days |
| 1900..S. | .. Netley .. | " .. | " .. | 5 .. |

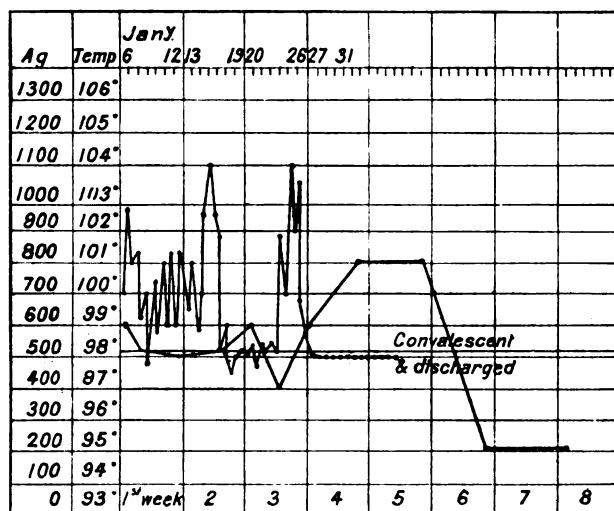
The agglutination of the micrococcus when brought in contact with dilutions of blood of patients suffering from Mediterranean fever was early established, and became an efficient method for diagnosing this at times most irregular disease, and years of experience at Malta, Netley and Haslar have proved its value, and, as in typhoid, serum diagnosis is now regularly carried out, the agglutinating properties are even more marked than in the latter disease, commencing earlier (five days Surgeon S.) and lasting long (one and a half years, B. S.), and acting in higher dilutions of serum, the clumping being more compact, and as seen macroscopically, more definite.

In September, 1899, C. Birt and G. Lamb made a number of observations showing the relative agglutinating powers of the serum during the course of the disease in 15 cases.¹

In repeating like experiments I wished to see whether their results would be confirmed.

The technique employed consisted in using an emulsion made from a five-day-old agar culture of the micrococcus in saline solution; this was drawn up into sterile pipettes and killed by heating for a period of fifteen minutes at 65° C., the same series of pipettes being utilised throughout in the cases. The blood was centrifugalised and the serum generally employed a few hours after it was obtained; the dilutions were made with a measuring pipette as recommended by Wright, an equal part of diluted serum (20 c.cm.) and emulsion being always used, so that the number of organisms present in each sedimentation tube was approximately the same in each instance; these were left at room temperature for twenty-four hours and the results then recorded, the dilutions ranging from 1 in 20 to 1 in 2,000.

Examinations to show the relative amount of agglutinins present have been made in 56 cases, excluding those in which two only were recorded; these were made weekly, a series from the ward being taken together, and the whole technique regularly performed



No. 1. Group 1, short duration, high agglutinins.

period on that station, and having been invalided home arrive either fairly convalescent, or to run through a series of relapses often very acute, which may end in complete recovery, or produce such a permanent cachexia and loss of strength that it necessitates their being invalided out of the service.

The number of cases which have been available for systematic observation has therefore been abundant, and they have been utilised to furnish the observa-

by myself. From some of these, charts showing the fever curve and agglutination curve have been drawn up and reduced by photography, so as to bring the long periods covered within reasonable limits.

Few cases were received early enough to ascertain the reactions during the first weeks of the fever, and there was no fatal case in the series; in the only case where the examinations were made at the commencement a reaction as high as 1 in 600 was present on the fifth day. From a careful study of the clinical characters of the cases and the agglutination curves, we find:—

(1) That in cases of short duration and moderate severity the agglutinins are generally sustained high, 400 and upwards, falling when normal health is established, as Mr. S., R.N., No. 1.

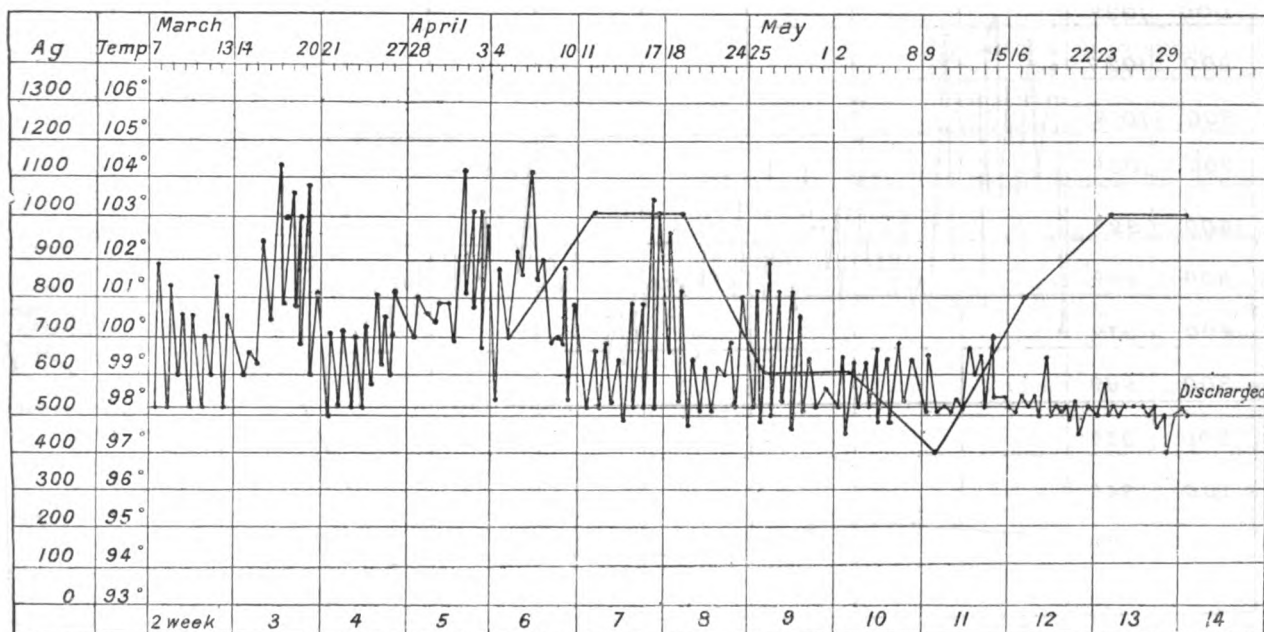
(2) Cases with acute clinical symptoms having at

first high agglutinins more or less regularly sustained, but generally falling as the fever passes off and debility becomes marked, to rise again in convalescence, as Mr. D., R.N., No. 2; G., No. 3.

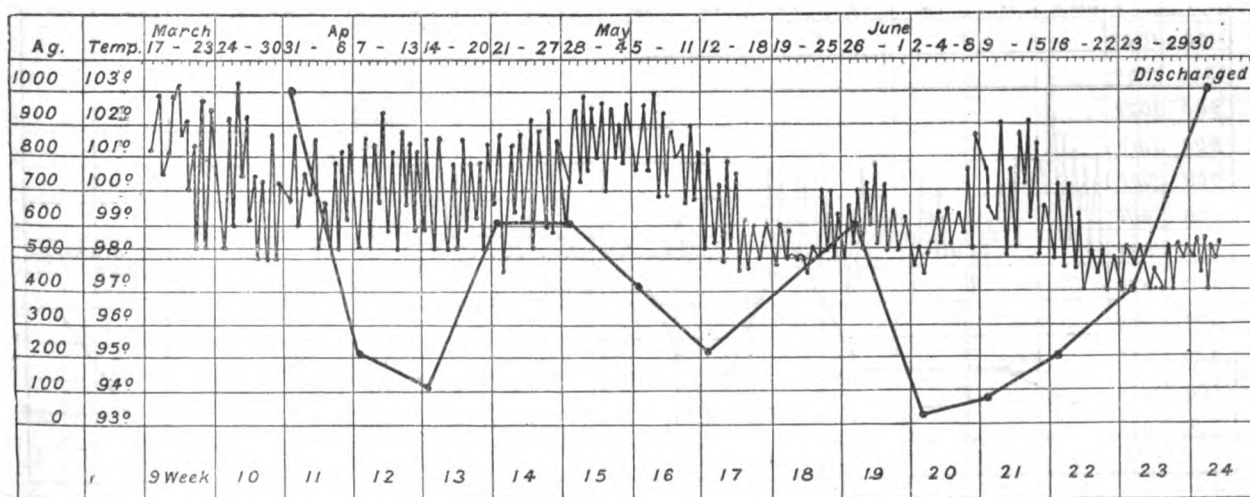
(3) Cases with acute symptoms, in which the agglutinins are from the first very low, rising more or less regularly as convalescence progresses; these cases are severe throughout. As R.W., No. 4, No. 5, No. 6.

(4) Cases with acute relapses where the agglutinins remain permanently low, and in which debility and anæmia are very great, many of these having to be finally invalided, as No. 7, No. 8, No. 9.

(5) Very chronic cases, with protracted slight relapses or continued fever of a mild hectic type, where from low agglutinins there is later a considerable increase, the patients ultimately making a good recovery, as No. 10, No. 11.



No. 2.—Group 2, longer case, acute symptoms, high agglutinins.



No. 3.—Group 2, long case, acute symptoms, high agglutinins.

(6) Very chronic cases, with prolonged irregular fever, great anæmia and debility, where the agglutinins remain constantly low and improvement is slight, most of these being invalided out of the service, as C., No. 12.

It is seen that the high agglutinating power of healthy convalescents slowly falls again, but reacting to 1 in 100 or less for months in debilitated convalescents; as seen above, there is no rise.

From the above, we may gather for prognosis that:—

(1) High sustained agglutinins in the early stages of the fever are favourable.

(2) A continuation of low agglutinins during the whole course of the fever and following cachexia is

bad, the cases dragging on for years with recurrent attacks of slight fever, and the neuroses so difficult to cure.

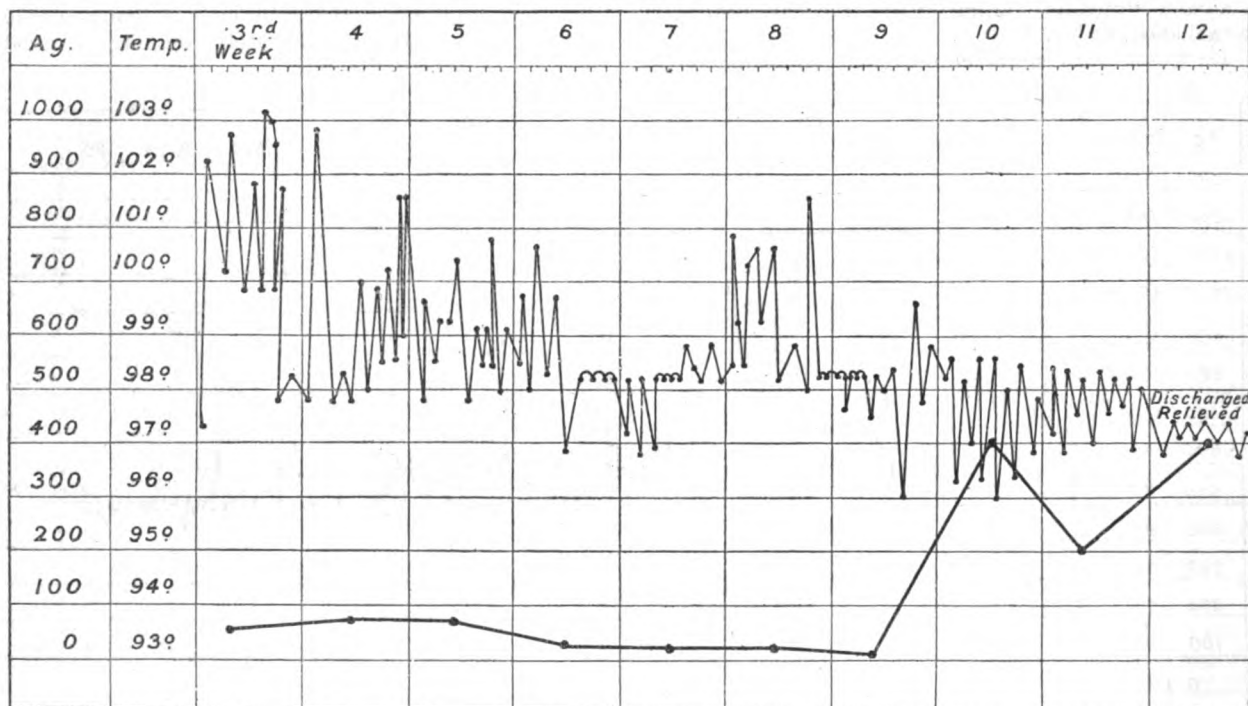
(3) A continuous rise, with improving clinical symptoms, indicates approaching convalescence.

(4) That as a rule there is no relation of the agglutination curve with that of the fever.

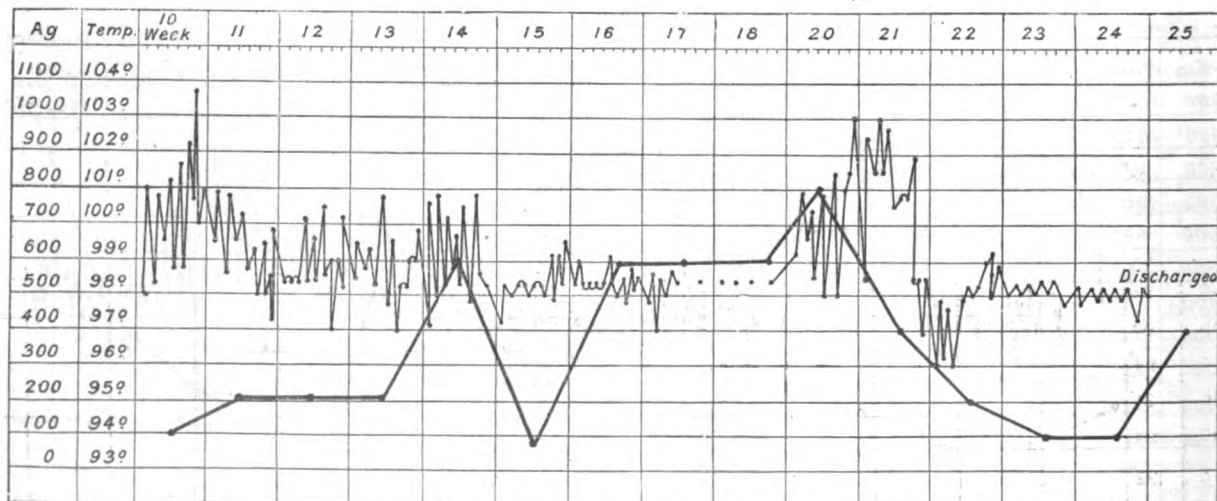
These observations are in harmony with those of Birt and Lamb.

Bactericidal Observations.

During this period, while investigating the characters of this disease in the cases invalided from the Mediterranean and elsewhere (for it is not restricted to the

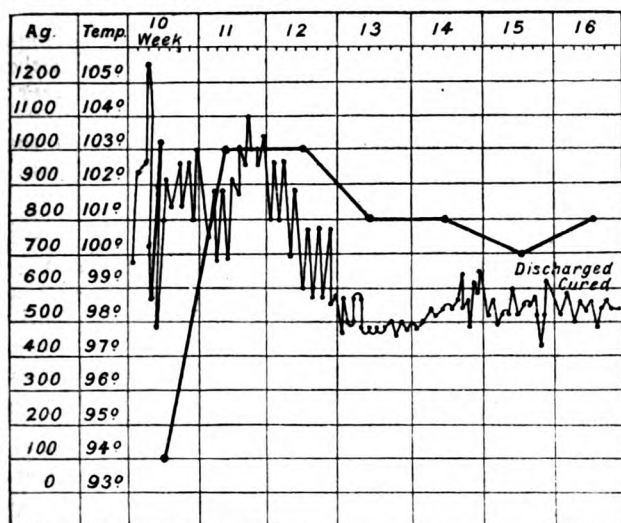


No. 4.—Group 3, moderately severe case, low agglutinins, rising in convalescence.



No. 5.—Group 3, severe case, low agglutinins, rising in convalescence.

former area, as has long been believed: quite lately I have examined blood from two men invalided from China who suffered from what clinically appeared to be this disease, or a fever closely allied, in which the serum gave strong agglutination reaction in dilutions 1:40, 60, 80, 100, and 200, but not above), the question of the presence of immune body in the blood becomes one of great importance; I have therefore carried out a series of observations to test the bacteri-



No. 6.—Group 3, severe case, low agglutinins, rising in convalescence.

cidal properties of fresh serum, which may be divided under the following headings:—

- (1) Those suffering from the fever at the time.
- (2) Those convalescent from an attack.
- (3) Those in health never having had an attack.
- (4) To see if there was any relationship between the bactericidal power and the agglutinating properties of the serum.

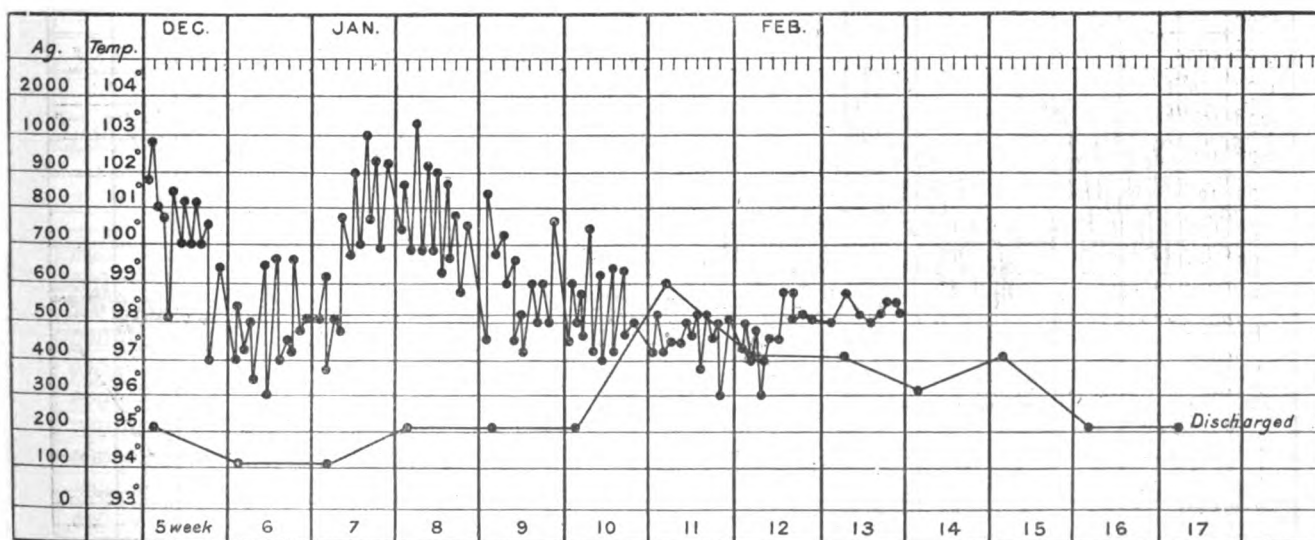
Technique employed: The finger having been well sterilised, a considerable quantity of blood is drawn

into sterile pipettes, and placed in the incubator at 37° C. for one to two hours, then centrifugalised. A broth culture of micrococcus melitensis of generally one week's growth is taken and dilutions made (1) 10 c.mm. are added to 10 c.cm. of broth and well mixed, and from this 10 c.mm. again to 10 c.cm. of broth, giving a dilution of 1 in 1,000,000 = A, and from this in carefully sterilised watch glasses dilutions of 1 in 10,000,000 = B, and 1 in 100,000,000 = C, are easily made; from these high dilutions sloped agar tubes are inoculated with 5 and 10 c.mm. and in four or five days the colonies counted, when the relative number of organisms in each dilution can be estimated.

Equal parts of clear blood serum and A, B, and C are drawn up in tubes with spiral twist as recommended by Wright,³ incubated at 37° C. for twenty-four hours, and then blown out on thick agar plates, which are incubated for three to seven days, when the presence or absence of growth of micrococcus melitensis can be observed; if there is no growth one infers that the serum has been able to kill off the organisms present in that dilution, and so estimate the bactericidal power, which is in all cases very considerably less than that found against the typhoid organism.

The following is a tabulated result of 15 cases so treated:—

From the above one would gather that in acute, chronic, and convalescent cases, the bactericidal power of the serum against the specific organism of Mediterranean fever is very slight, very much less than that usually of healthy individuals who have never suffered from the disease, and that if this procedure gives any adequate estimation of the immunity of the subject, then those who have lately recovered from the disease are less able to resist the invasion of the organism if introduced, and therefore more prone for a period to reinfection, and that if this is so, men who have lately recovered from an attack should not return to the endemic area for some time, and also the necessity for early invaliding becomes more apparent.



No. 7.—Group 4, severe case, great anaemia, incomplete recovery, low agglutinins throughout.

RESULT OF BACTERICIDAL OBSERVATIONS.

| Case | Name | Character | Date | No. of Organisms | Result | Control | Result |
|------|------|------------------|----------|------------------|------------|---------------------|-------------|
| 1 | D. | Acute attack | April 28 | 2 in 5 c.mm. | Not Killed | Healthy labourer | Killed. |
| | | Convalescent | May 28 | 1 " 5 " | " | Healthy surgeon (1) | Not killed. |
| 2 | T. | Acute attack | " 18 | 8 " 5 " | " | " (2) | Killed. |
| | " | " | June 8 | 1 " 10 " | " | " (3) | " |
| 3 | H. | " | May 11 | 8 " 5 " | " | " (1) | " |
| | " | " | " 18 | 1 " 5 " | " | " (1) | Not killed. |
| | " | " | " 25 | 1 " 5 " | " | " (1) | " |
| | " | Convalescent | June 22 | 1 " 5 " | " | " (5) | Killed. |
| 4 | S. | Acute attack | May 25 | 1 " 5 " | " | " (4) | Not killed. |
| | " | " | June 8 | 1 " 10 " | " | " (3) | Killed. |
| 5 | O. | " | May 11 | 1 " 5 " | " | " (1) | Not killed. |
| 6 | W. | " | June 8 | 1 " 5 " | " | " (3) | Killed. |
| 7 | F. | Chronic cachexia | " 8 | 1 " 5 " | " | " (3) | " |
| 8 | Q. | " | " 22 | 1 " 5 " | " | " (5) | " |
| 9 | M. | " | May 18 | 1 " 5 " | " | " (1) | Not killed. |
| | " | " | " 25 | 1 " 5 " | " | " (4) | " |
| | " | " | June 12 | 1 " 5 " | " | " (5) | Killed. |
| 10 | S. | " | May 6 | 1 " 5 " | " | " (3) | " |
| | " | " | " 18 | 1 " 5 " | " | " (1) | Not killed. |
| 11 | C. | " | April 29 | 3 " 5 " | " | " (5) | " |
| 12 | B. | Convalescent | May 6 | 1 " 5 " | " | " (3) | Killed. |
| 13 | B. | " | " 6 | 1 " 5 " | " | " (3) | " |
| 14 | S. | " | " 6 | 1 " 5 " | Killed | " (3) | " |
| 15 | T. | " | " 6 | 1 " 5 " | Not killed | " (3) | " |

any marked increase or diminution in activity of the leucocytes. The method of Leishman was followed strictly, but the difficulties of enumeration are considerable, owing to the minute size of the micrococcus, his modified Romanowski's stain giving the best results, though for ordinary blood work I prefer that of Louis Jenner, being more rapid, cleaner, and less trouble if the slides are placed upright in a jar of the stain.

In thirteen cases examined the average organisms found inside the polymorphonuclear leucocytes were few in cases of Mediterranean fever, whereas in the controls of normal blood they were always higher; it would therefore seem that not only is the bactericidal power of the blood low, but that also the phagocytic properties of the leucocytes is diminished.

General Blood Changes.

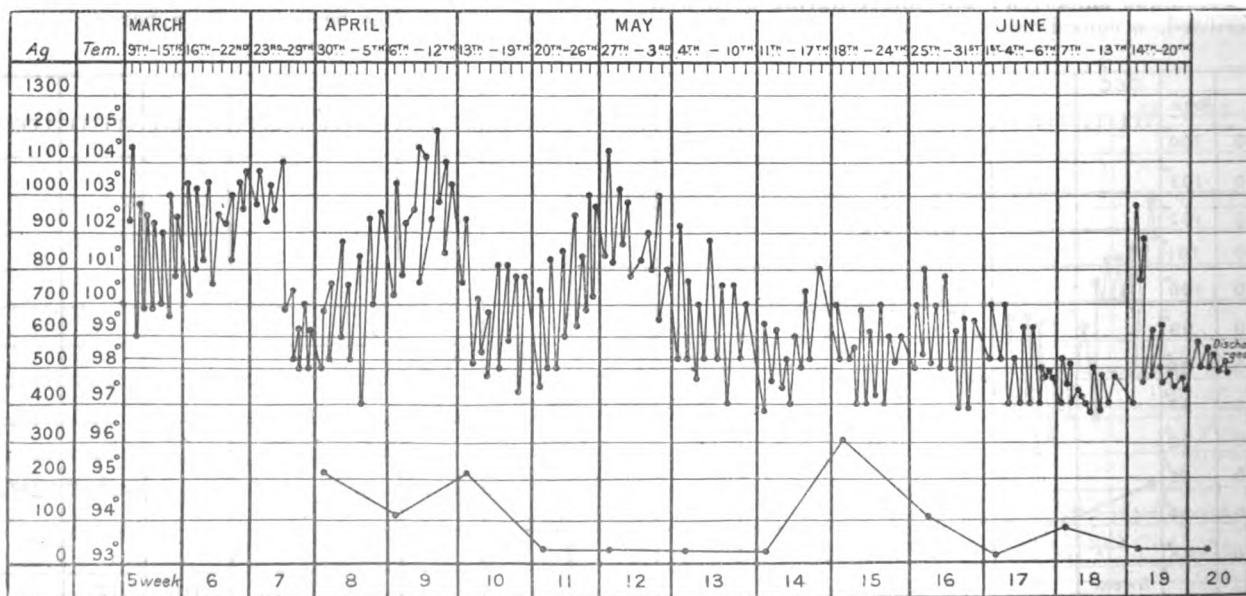
In the weekly examinations of the blood the hydræmic condition of the fluid is very apparent to every one. The red cells often falling to a very low figure, in one counted as little as 2,800,000 frequently between 3,000,000 and 4,000,000, the cells themselves being deficient in hæmoglobin, very irregular in size and shape, microcytes being particularly abundant, but I have never found any nucleated red forms in the routine examination of the blood films, though blood plates in the more advanced cachectic cases are certainly increased, staining readily with Louis Jenner's method. The white cells are often relatively increased, but never, I believe, absolutely, the highest count being 6,600, but there is undoubtedly in most instances a great relative preponderance of the mononuclear basophilic cells, sometimes exceeding in number the neutrophils or polymorphonuclear leucocytes, the proportion of the former ranging from 35 to 76 per cent., while that of normal blood is 25 to 35 per cent., and in leucocytosis from pus or acute pneumonia it is 11 to 14 per cent.

This great diminution in the number of red cells

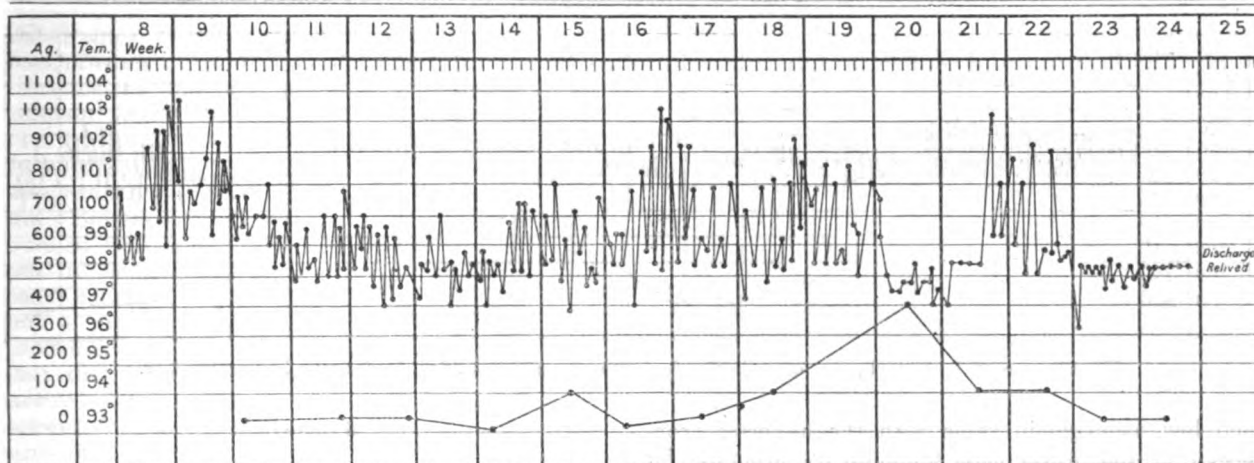
Phagocytic Power of Leucocytes.

Besides, however, the protective influence of the immunising bodies in the blood serum, we have also the active phagocytic powers of the leucocytes themselves in carrying out the warfare against these minute organisms, a principle brought forward strongly by Metchnikoff to explain the facts of natural and acquired immunity.

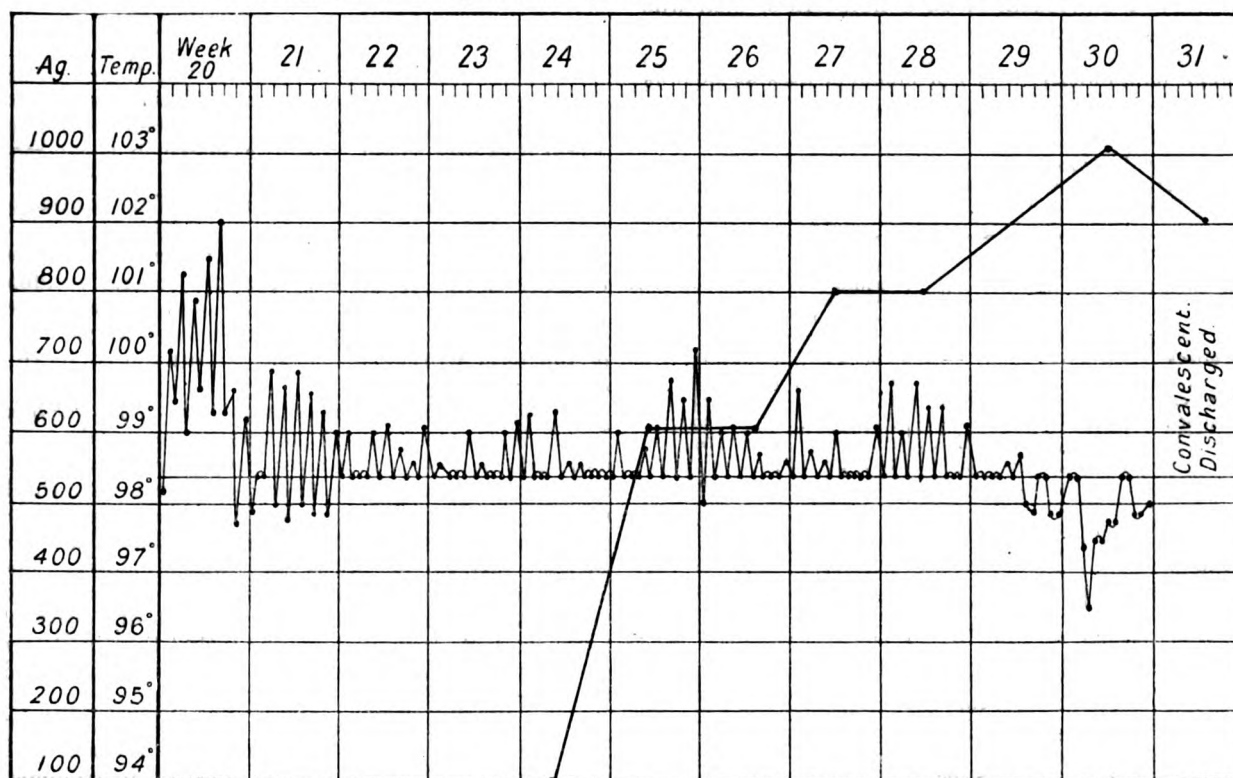
The technique for demonstrating this has been lately described by Major Leishman,⁸ it was interesting and important to see whether in this disease there was



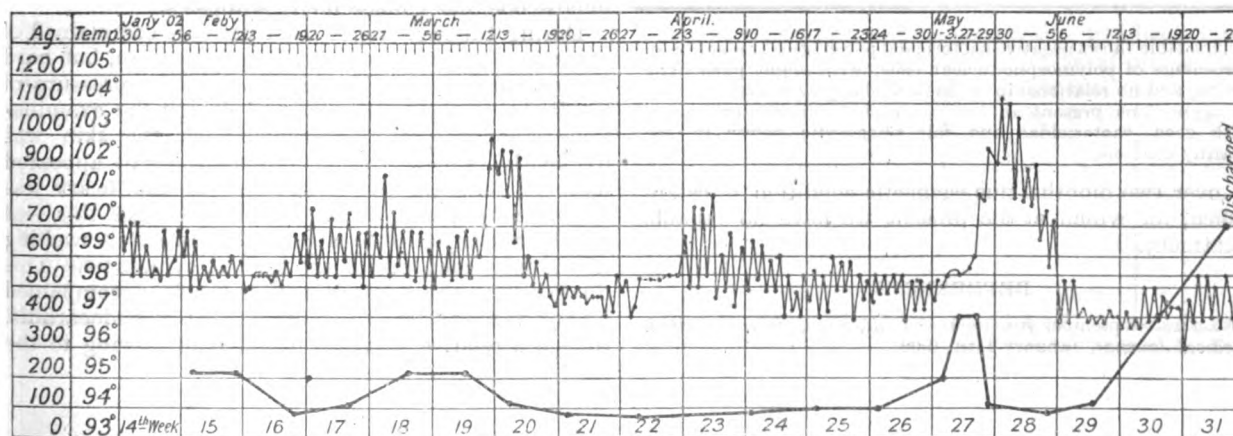
No. 8.—Group 4, severe case, great anemia, incomplete recovery, low agglutinins throughout.



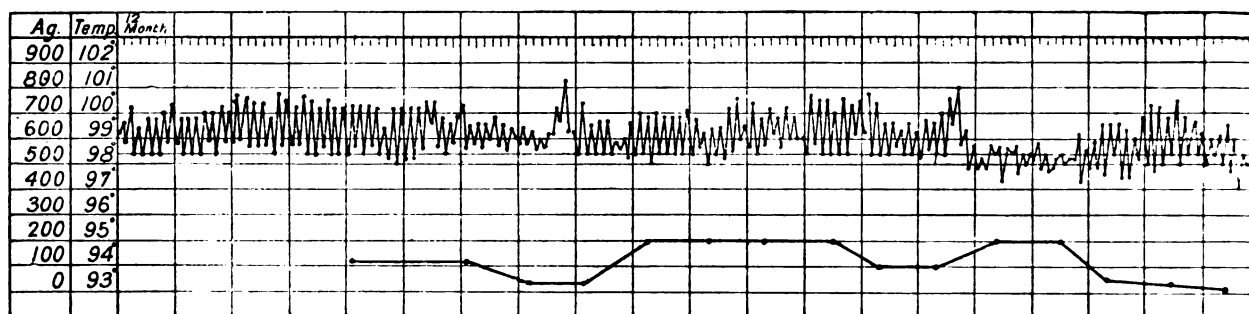
No. 9.—Group 4, severe case, great anæmia, incomplete recovery, low agglutinins throughout.



No. 10.—Group 5, chronic case, good recovery, agglutinins rising, high in convalescence.



No. 11.—Group 5, chronic case, fair recovery, persistently low agglutinins, rising before discharge.



No. 12.—Group 6, very chronic case, hectic fever (two years), invalided, persistently low agglutinins.

and low hæmoglobin value with the abnormal condition of the white cells undoubtedly accounts for the intense cachexia, slow, and so often incomplete, recovery of unfortunately such numbers of men who have returned to England suffering from Mediterranean fever; and I believe strongly that all cases should be removed from the endemic area as soon as possible, for if the disease is protracted for a period

TABLE GIVING THE RELATION OF THE BACTERICIDAL, PHAGOCYtic, AND AGGLUTINATIVE POWERS, WITH PERCENTAGE OF MONONUCLEAR CELLS TO TOTAL WHITE CELLS.

| No. | Character of Case | Bactericidal Power | Phagocytic Power | Highest Diminution with Complete Agglutination | Relative Percentage of Mononuclear Cells to Total Leucocytes |
|-----|------------------------|--------------------|------------------|--|--|
| 1 | Acute attack ... | Nil ... | — | I in 40 ... | 75 |
| | " " " " ... | " " " " ... | Fair ... | I ,, 400 ... | 65 |
| | Convalescent ... | Good ... | Slight | I ,, 40 ... | 53 |
| 2 | Acute attack ... | Nil ... | — | I ,, 100 ... | 59 |
| | " " " " ... | " " " " ... | Fair ... | I ,, 400 ... | 64 |
| 3 | " " " " ... | " " " " ... | " " " " ... | I ,, 40 ... | |
| 4 | " " " " ... | " " " " ... | Slight | I ,, 800 ... | 46, short duration. |
| 5 | Chronic cachexia ... | " " " " ... | " " " " ... | I ,, 40 ... | 46 |
| 6 | " " " " ... | " " " " ... | " " " " ... | I ,, 100 ... | |
| | Convalescent ... | " " " " ... | " " " " ... | I ,, 800 ... | 57 |
| 7 | Chronic cachexia ... | " " " " ... | " " " " ... | I ,, 400 ... | 69 |
| 8 | " " " " ... | " " " " ... | Nil | I ,, 100 ... | |
| 9 | Convalescent ... | " " " " ... | Slight | I ,, 100 ... | 26, slight anæmia. |
| 10 | " " " " ... | " " " " ... | " " " " ... | I ,, 100 ... | 59 |
| 11 | " " " " ... | " " " " ... | " " " " ... | I ,, 400 ... | 41 |
| 12 | " " " " ... | " " " " ... | Nil | I ,, 100 ... | 44 |
| 13 | " " " " 1½ yr. ... | Fair ... | Good ... | I ,, 40 ... | 40 |
| 14 | Healthy control, 1 ... | Good ... | — | Nil ... | 33 |
| 15 | " " " " 2 ... | " " " " ... | Slight | " " " " ... | 31 |
| 16 | " " " " 3 ... | Nil ... | Fair ... | " " " " ... | — |
| 17 | " " " " 4 ... | Good ... | " " " " ... | " " " " ... | 35 |

This table shows negative bactericidal power, great reduction in percentage of polymorphonuclear cells, with slight phagocytic action, and no relationship of bactericidal power with amount of agglutinins present in the cases of Mediterranean fever, with good bactericidal and fair phagocytic power in the healthy controls.

of over two months this cachectic condition is established, for which at the present we have no reliable treatment.

REFERENCES.

¹ *Lancet*, September 9th, 1899. ² *Ibid.*, June, 1901. ³ *British Medical Journal*, January 11th, 1902.

Reprints.

WHAT IS THE NATURE OF THE PORTO RICAN "ANÆMIA"?

By HERBERT M. McCONATHY, M.D.

DURING the years of 1899 and 1900 the attention of the U.S. Army surgeons who were then serving in Porto Rico was called to a disease which is common among the inhabitants of that island, and which is known there simply as "anæmia." This disease is interesting first, on account of its high mortality—there are practically no recoveries; and secondly, on account of its prevalence. I asked several resident Spanish physicians for an estimate as to its prevalence, and was astonished to find that at least 50 per cent. of the total number of deaths are attributed to this disease alone.

Opinions as to the cause of this trouble are various. Insufficient nourishment is, naturally, the usual reason assigned, because the poorer people live almost exclusively on plantains. There are some who think rheumatism an important factor on account of the pains in the limbs during the earlier stages and the frequent involvement of the heart which follows. The only real study of the disease of which I have heard was that made by Lieut. Bailey K. Ashford, Assistant Surgeon, U.S. Army, who was at that time in charge of the hospital at Ponce. Dr. Ashford pronounced the disease ankylostomiasis.

From about September 1st, 1899, to about August 1st, 1900, I was stationed in Adjuntas, a small town in the interior where this disease is especially common, and during these eleven months I saw hundreds of cases. As my post was small I could not secure a microscope, but I made many autopsies.

During the first stages most patients complained of pains in the limbs, sometimes quite severe, but hard to locate definitely. Tenderness on deep pressure of the limbs was generally elicited during the examination although partial anæsthesia of the skin was frequently noted. In many cases the gait appeared more or less ataxic. Dilatation of the heart was always found, and during the later stages this was usually accompanied by a general anasarca; a swelling of the feet being one of the earliest symptoms. This dilatation of the heart is not, as a rule, accompanied by any valvular disease or other signs of endocarditis, though a relative valvular insufficiency, owing to the

dilated rings, is very early and constantly noted, and gives, of course, a strong systolic murmur.

Insufficient and improper food is, without doubt, the main predisposing cause of the disease, in fact it is hard to understand how a human being can sustain life on the diet on which a majority of the Porto Ricans subsist, especially those who live in the mountain. But starvation cannot be the only cause of this so-called "anæmia," for cases are occasionally met with in persons who are well fed. The rheumatism theory can be dropped on account of the absence of definite joint symptoms and of endo- or pericarditis. As to ankylostomiasis, I can readily credit the statement that it is wide-spread in Porto Rico and productive of much harm, but this diagnosis will not account for the partial anæsthesia, the rheumatic pains, and the tenderness of the muscles on deep pressure. The dilatation of the heart I found to be such an early and constant symptom that it is hard for me to believe it merely a result of the anæmia.

Since coming to the Philippines I have seen a disease which reminds me very forcibly of the Porto Rican one; it is beri-beri. The more I see of beri-beri the more striking the resemblance seems. I regret that I did not test the knee-jerk in the Porto Rican "anæmia," for the absence of this reflex is an important point in the diagnosis of beri-beri, and this is the only thing lacking to establish, in my opinion, the identity of the two diseases. It would not surprise me if it were found upon investigation that beri-beri and ankylostomiasis were combined in many of these cases of Porto Rican "anæmia."

This disease is generally chronic in its course, and its victims exist in a state of invalidism or semi-invalidism for months before they finally succumb. This fact, combined with the exceeding prevalence of the malady, greatly impairs the working power of the population and interferes most seriously with the progress of the island.—Reprinted from the "*Journal of the Association of Military Surgeons of the U.S.*," May, 1902.

WE note with satisfaction the following honours recently conferred by the King:—

Order of the Bath.

Brigade Surgeon-Lieutenant-Colonel A. Crombie, M.D., retired, late Indian Medical Service, to be Companion of the Order of the Bath, Civil Division.

Order of St. Michael and St. George.

William H. W. Strachan, Chief Medical Officer of the Colony of Lagos, to be Companion of the Order of St. Michael and St. George.

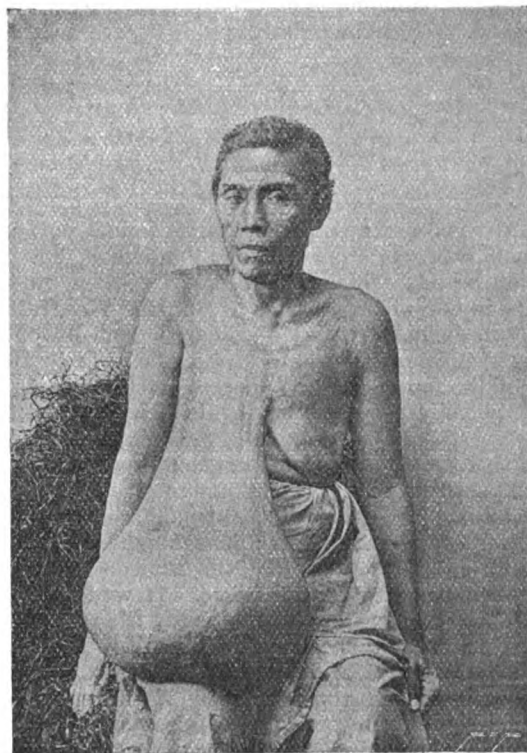
Obituary.

It is with much regret that we have to record the death of Stanford Harris, M.D.Durh., M.R.C.S.Eng., L.S.A., which took place recently in Southampton. Dr. Harris was the Medical Superintendent of the Guimar Hospital for tuberculosis, at Teneriffe, and one of the pioneers of the open-air and climatic treatment of consumption.

Reviews.

THE DISEASES OF THE SAMOAN ISLANDS.

Dr. Augustin Krümer, of the German Navy, in an addendum to the second volume of reports published in Stuttgart by E. Nägele, describes the most important skin diseases met with on the South Sea Islands. Amongst these he refers to *tinea circinata* and im-



bricata, verruga and framboesia, elephantiasis and leprosy. The descriptions are good, and Dr. Krümer's long experience in Samoa renders the statements authentic. The book is illustrated by photographs taken by himself, one of which—elephantiasis of the mamma—we reproduce by the permission of the publisher.

TROPICAL HYGIENE (Tropen Hygiene). By Professor Dr. F. Plehn. With 5 plates and 5 illustrations in the text. 1902: Gustav Fischer, Jena. Pp. 282.

Dr. F. Plehn systematically divides his book into twenty lectures, comprising:—(1) Tropical climate in general. (2) The climate in the tropical colonies of Germany. (3) The influence of the tropical climate on the human organism, and acclimatisation. (4) Tropical malaria. (5) The excitors of malaria and manner of transmission. (6) The prevention of malaria. (7) Course and treatment of tropical malaria. (8) Blackwater fever. (9) Small-pox and plague in regard to the German Colonies. (10) Tropical skin diseases. (11) Gastric and intestinal diseases in the Tropics. (12) Animal parasites of man in the Tropics. (13) Snake venom and arrow poison in the

Tropics. (14) Diseases of eyes and ears; injuries. (15) Preparation for Colonial service and voyage out. (16) Tropical houses. (17) Position of stations in the Tropics. (18) Tropical life on the station. (19) Hygiene of expeditions. (20) Drugs for the Tropics.

The book is convenient in size, clear in style, and should prove of the greatest service to laymen as well as medical men proceeding from German-speaking countries to warm countries.

All Dr. F. Plehn's work is so thorough and reliable that implicit confidence may be placed on his statements and his recommendations.

News and Notes.

BRANDY FROM THE GRAPE.—For medicinal purposes we would welcome a pure brandy. There is but one form of brandy which can be so regarded, and it remains in our pharmacopœia as *Spiritus Vini Gallici*, B.P., and is defined as "a coloured and flavoured variety of alcohol distilled from French wine." How much of the brandy on the market, even when stated to be specially prepared for invalids, has any grape product in its composition, it is difficult to ascertain. In North Germany, Holland, and this country, brandy is produced from raw grain or potatoes, consisting of spirit and flavourings wholly independent of the grape. That such compounds should masquerade as brandy according to the definition of the *Pharmacopœia* is, to say the least of it, unfortunate. It would appear that it does not pay to produce brandy from the grape in Europe. Why this is so it is difficult to ascertain, for grapes and to spare seem plentiful enough. We must look beyond Europe, evidently, if we are to obtain a brandy made from this grape juice; to a country where the grape is not wholly used for the production of still or sparkling wines, but where there is a surplus from which brandy may be distilled. The Australians are making an effort to once more place brandy made from the grape on the market in Britain, and we have been favoured with samples of the "Orion" brandy professing to be made from the grape. The aroma of this brandy leaves no doubt that the producers' statement is correct, and could they assure the medical profession that all their brandies are made from the grape they would command an extensive sale.

THE LADY CURZON'S HOSPITAL AT SECUNDERABAD.—The new Maternity ward which has been built and presented by Sett Ram Gopal, as an addition to the Civil Hospital, was opened on September 5th, 1902. The new Maternity ward provides accommodation for 14 beds—6 for Europeans and 8 for natives. The total number of females admitted for confinement in the other wards last year was 100.

RESEARCH LABORATORIES AT KHARTOUM—PRESENTATION BY MR. HENRY S. WELLCOME.—The Gordon Memorial College at Khartoum which Lord Kitchener opened on Saturday last, is now ready for the chemical and bacteriological research laboratories

presented by Mr. Henry S. Wellcome during his recent visit to the Soudan. The fixtures and appliances made in England have already been shipped. The equipment for scientific work is most complete in every detail, and will be equal to any similar laboratories in Europe. The Sirdar has appointed as Director of these research laboratories, Andrew Balfour, M.D., B.Sc., D.P.H., of Edinburgh, whose work in bacteriology is well known in professional circles. The Soudan presents exceptional opportunities for the study of tropical diseases, especially malaria, typhoid and dysentery, and it is anticipated that the results of the investigations of Dr. Balfour and his staff will be of the greatest importance. Dr. Balfour will also assist the authorities in the investigation of the criminal poisoning cases, which are very frequent in the Soudan. The character of the poisons used by the natives is at present often obscure, and it is possible that the work in these laboratories may considerably increase our knowledge of toxic agents. Apart from the original researches and general sanitary work, Dr. Balfour and his staff will devote their attention to the study of the cereals, textile fibres, and various matters affecting the development of the agricultural and mineral resources of the country.

Current Literature.

SUMMER DIARRHŒA IN CHILDREN—TREATMENT.

(1) W. L. Harris concludes that summer diarrhœa is to a great extent a preventable disease, and it is our duty to do all we can to instruct the mother and nurse in the care and feeding of children. In our treatment of this disease we should always stop the milk, give a purge, and then carefully regulate the diet for a few days, even in the simplest and mildest cases, and in this way prevent many a case of the more serious forms of diarrhœa. The treatment of the first few days is the most important in all cases, and even the simplest cases should never be neglected under the delusion that it is natural for a teething child to have diarrhœa.

(2) Maurice Ostheimer, after considering the prophylaxis of the disease, states that the most important treatment is absolute withdrawal of food, no matter what the infant is taking. The child should be kept in its carriage out of doors, or if this is not possible, in the largest room in the house with the windows open. Few drugs are necessary. In case of vomiting, calomel is given. When there is no vomiting, castor oil is indicated. If frequent bowel movements persist, bismuth subnitrate with a little salol is given. During convalescence, a few drops of tincture of *nux vomica* will often be of service. As the Health Boards of the large cities continue to distribute hygienic regulations for the care of babies, the poor are gradually learning how to care for their children.

(3) James H. McKee offers the following classification of these diarrhœas:—(1.) Dyspeptic; due chiefly to improper or to imperfectly-digested food. Bacteria

may or may not play a part; acute intestinal indigestion; dyspeptic diarrhoea. (ii.) Infectious; in which bacteria of different kinds play the important rôle. Such organisms are almost invariably introduced in food, and nearly always in cow's milk. (a) Fermentative diarrhoea; mild gastro-enteric infection. (b) Ileocolitis, enterocolitis, acute or chronic. (c) Cholera infantum. The first consideration in any form of diarrhoea is the diet. In the case of a bottle-fed baby, unless the trouble is mild dyspeptic diarrhoea, it is safer to withdraw milk. In dyspeptic diarrhoea of mild grade, the weaker milk formula or peptonised milk is advisable. In case of a milk infection, milk is withdrawn. Boiled water should be given frequently for the first twenty-four hours, but no nourishment. Barley water may then be given. If the baby will not take it a few drops of aromatic spirits of ammonia will often make it acceptable. When the diarrhoea has been caused by an excess of carbohydrates, barley water is contraindicated. Beef juice is usually well borne. Animal broths may be given on the third day. Milk feeding should always be gradually resumed. A purge should always be given as a preliminary. Castor oil and calomel are the best. In some cases an astringent, such as the salts of bismuth, or silver nitrate, is indicated. The writer has had good results from salol as an intestinal antiseptic. Some form of opium is indicated in certain cases. Enteroclysis and hypodermoclysis are indicated in some cases. As stimulants, when necessary, brandy, old whiskey, atropine and strychnine are valuable. The cold pack is valuable in the treatment of fever. The most frequent complication is catarrhal pneumonia, which is to be treated as it is when occurring under ordinary conditions.—*Philadelphia Med. Journ.*, July 26th, 1902.

(4) Dr. Loms Fischer points out that in the presence of fever, gastro-enteric disorders, summer diarrhoea, &c., in children, it is often requisite to stop milk feeding. Under such circumstances the following substitutes have been of special value in Dr. Fischer's hands, and are offered for temporary use in gastric and intestinal derangements.

Formulae for weak infants in substitute feeding:—

When vomiting and diarrhoea persist give either

Barley water 4 ounces

Or,

Oatmeal water 4 ounces

Feed the child with this amount every two or three hours.

Sweeten with granulated sugar, half a teaspoonful to each bottle; or, if fermentation exists, as shown by colic, greenish stools, and eructations, with half a grain of saccharine in place of the sugar.

The following are Dr. Fischer's methods of preparing barley water and oatmeal water:—

Barley water can best be made by taking one heaping tablespoonful of ground barley flour and adding the same to one pint of water. Boil this thoroughly for half an hour, then strain through cheese-cloth and add enough water to make one pint of barley water. When barley water is given for any length of time, and constipation results therefrom, then glycerine should be added. One teaspoonful of glycerine to each teacupful of barley water will make the same quite palatable, and will offset the constipating tendency.

Oatmeal water can be made by adding one tablespoonful of oatmeal flour to a pint of water, boiling the same in the same manner above described for the preparation of barley water.

If the child is underfed, then frequently the addition of the white of a raw egg, well beaten, with either the barley water or oatmeal water, will be found advantageous.

Dr. Fischer has also frequently added the yolk of an egg, well beaten, with barley water or oatmeal water properly sweetened, as a temporary substitute.

Dr. Fischer's formula for almond milk food is:—

Almond milk 4 ounces
Granulated sugar... .. 1 teaspoonful

M. Give the foregoing quantity every three hours.

Formulae for whey feeding:—

To make whey, take half a pint of fresh milk, heated lukewarm, not warmer than can be agreeably borne by the mouth (about 115° F.); add one teaspoonful of essence of pepsine, and stir just enough to mix. Pour into custard cups; let it stand until firmly curdled; then beat up with a fork until the curd is finely divided; now strain and the whey is ready for use.

The whey may be administered as follows:—

For an infant under six months:—

Whey 2 ounces
Milk 1 ounce

Warm to blood temperature (about 100° F.) for three minutes, then feed. The above quantity can be given every two hours or two hours and a half.

When feeding a child from two to four months old:—

Whey 2 ounces
Milk 2 ounces

To be given every three hours.

If the above is well borne, we must gradually increase by adding an ounce of food; the formula will then be:—

Whey 2½ ounces
Milk 2½ ounces

Give the above quantity every three hours.

The general condition of the infant—its sleep, its stool, and its body weight—is the important factor to determine an increase in the quantity of food.

If the child cries very much after feeding and appears hungry, then we may give:—

Whey 3 ounces
Milk 3 ounces

Every three hours.

Some children at three months will take very readily six ounces of food. If the appetite warrants it, and the stool is homogeneous and well digested, then we need not hesitate to give the following:—

Whey 3 ounces
Milk 4 ounces

Every three hours.

The weight is the determining factor. If the child does not thrive, increase the quantity of milk and decrease the whey.

In the case of feeble and debilitated children, Dr. Fischer frequently orders sweetened whey instead of water for quenching thirst. This is especially valuable in summer.—*Med. Record*, August 2nd, 1902.

TREATMENT OF DYSENTERY.—J. B. Brandon, M.D., of Dudley, Mo. (*Medical Brief*, October, 1902), recommends the following mixture, to be used when the bowels move very frequently, when there is much pain, when the evacuations consist only of blood and mucus, and the tongue is of a scarlet colour.

R Sulph. Acid. Arom. or Dil. 3ss.
Tinct. Opil 3ss.
Sat. Sol. Magnesia Sulph. q. s. ad 3iii.

Of this mixture a teaspoonful is to be given every two or three hours in water, until the blood and mucus has disappeared from the stool, which generally occurs in between twelve to forty-eight hours, according to the severity of the attack.

Dr. Brandon, in addition, advises the local application of turpentine to the abdomen. The diet must be light, consisting by preference of rice, soup, and chicken broth, given in small quantities at frequent intervals. When the blood has disappeared from the stools, the usual lactopeptin, pepsin and bismuth are given in doses.

In cases with symptoms as follows: Bowels moving freely, tenderness in the bowels, slight rise of temperature— 101° — 102.5° F., with the tongue heavily coated, and edges of tongue strawberry colour, the following mixture should be given at once:—

R Hyd. Subchlor. 2 grs.
Sodii Bicarb. 2 „
Salol 24 „
Bismuth Subnit. 32 „

M. et ft. Sig.: One powder every three hours until the tongue cleans.

(Alternate this with):—

R Bismuth 3ii.
Elix. lactopeptin q. s. ad 3ii.

M. Sig.: Every three hours.

PLAGUE.

INDIA.—During the two weeks ending October 11th and 18th, the deaths from plague in India numbered 10,108 and 10,750 respectively; of these totals, 7,057 and 7,351 respectively occurred in the Bombay districts.

EGYPT.—No fresh cases of plague occurred in Egypt during the week ending October 19th.

SOUTH AFRICA.—During the week ending October 4th, no cases of plague were reported in human beings from any part of Cape Colony, but at Port Elizabeth plague-infected rats were found.

MAURITIUS.—During the week ending October 30th, 29 fresh cases of plague and 16 deaths from the disease occurred in Mauritius.

CHOLERA.

EGYPT.—During the week ending October 20th, 685 cases of cholera occurred in Egypt, against 819 in the previous week; of these cases 15 occurred in Cairo and 60 in Alexandria.

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Archives Russes de Pathologie, de Médec. Clinique et de Bacteriologie.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.

Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Brooklyn Medical Journal.
Caducée.
Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Esercito.
Hong Kong Telegraph.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
Journal of the American Medical Association.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Medical Record.
Medical Review.
Merck's Archives.
New York Medical Journal.
New York Post-Graduate.
Pacific Medical Journal.
Polyclinic.
Public Health.
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- 4.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.
- 5.—Correspondents should look for replies under the heading "Answers to Correspondents."

The Journal of Tropical Medicine.

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Original Communication.

ADULT FORM OF FILARIA DEMARQUAI.

By C. W. DANIELS, M.B., M.R.C.S.

Two specimens of female adults of the filarial embryos known as *Filaria Demarquai* were presented to the London School of Tropical Medicine by Dr. Galgey, of St. Lucia.

He found them in the body of a native of St. Lucia in the connective tissue of the mesentery. There were five worms found, all females. Two of the other specimens have also been examined by me. These worms present the usual appearances of the human filaria whose embryos circulate in the blood. They are long, thin worms, with an unarmed head and terminal mouth. The genital opening is near the head and is single. There are two ovarian tubes terminating together in a pouch-like uterus. The alimentary canal is nearly straight and terminates in an anus which is subterminal. The opening of the anus is marked by a slight papilla.

The worms were fairly uniform in most of their measurements and in the shape of the cephalic and caudal extremities, and were sexually mature. In all the specimens there was the marked cuticular thickening covering the tip of the tail.

The comparative measurement of these female adults and of those of *F. Bancrofti*, *F. perstans*, and *F. Ozzardi*, are given in the subjoined table.

It will be seen from this table that *Filaria Demarquai* is a thicker worm than *Filaria perstans*. It differs from *Filaria Bancrofti* and *Filaria Ozzardi* in the greater size of the head, in the smaller tail, and particularly in the marked cuticular thickening at the tip of the tail. This thickening is knobby, but the divisions are not so marked as in *Filaria perstans*. This cuticular thickening in other worms does not vary in life. In such other worms as have it, as

Filaria perstans and some of the bird filaria, it is constant, and it was present in each of the four specimens of Dr. Galgey's examined.

Some of the measurements of filaria, particularly those about the head, vary considerably in life, but as in these specimens the head has not got the globular form which is characteristic of the retracted head the measurements cannot be taken as indicating the extreme measurement.

| | ADULT FEMALES | | | |
|--|---|--|--|--|
| | <i>Filaria Perstans</i> | <i>Filaria Bancrofti</i> | <i>Filaria Ozzardi</i> | <i>Filaria Demarquai</i> |
| | Mm. | Mm. | Mm. | Mm. |
| Length .. | 70-80 | 85-90 | 81 | 65-80 |
| Greatest thickness .. | ·12 | ·2-·26 | ·21 | ·21-·25 |
| Diameter of head .. | ·07 | ·055 | ·05 | ·09-·1 |
| Distance of genital pore from head .. | ·6 | ·66-·75 | ·71 | ·76 |
| Diameter at genital pore .. | ·07 | ·14 | ·12 | ·1 |
| Distance from tail of anal papilla .. | ·145 | ·225 | ·23 | ·25 |
| Diameter at anal papilla .. | ·05 | ·1 | ·075 | ·07 |
| Character of tail .. | Curved. Double terminal cuticular thickenings | Bluntly truncated. No cuticular thickening | Bluntly truncated. Slightly bulbous. No cuticular thickening | Curved. Rapidly diminishes in size just below anal papilla. Cuticular thickening over tip of tail. |
| Diameter near tip of tail, before termination .. | ·02 | ·06 | ·045 | ·03 |

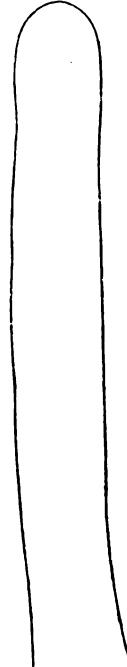
The specimens had been mounted by Dr. Galgey in glycerine jelly and were in excellent condition, showing no signs of distortion. There is a previous description* of this worm by Dr. Ozzard from a specimen received from Dr. Galgey, but as in that case the worm was much distorted the measurements were considered by Dr. Ozzard to be unreliable.



Head of *Filaria Bancrofti*, ♀.
(Dr. Ozzard's case.)



Head of *Filaria Ozzardi*, ♀.



Head of *Filaria Demarquati*, ♀.
(Dr. Galgey's case.)



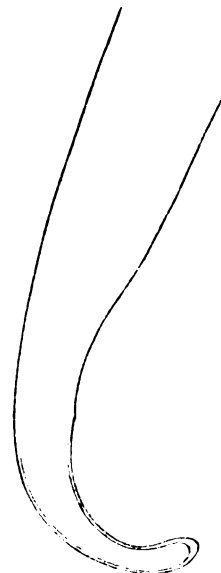
Head of *Filaria Perstans*, ♀.



Tail of *Filaria Bancrofti*, ♀.



Tail of *Filaria Ozzardi*, ♀.



Tail of *Filaria Demarquati*, ♀.



Tail of *Filaria Perstans*, ♀.

There can, I think, be no doubt that this is distinct from *Filaria Ozzardi*, though the embryos of the two are indistinguishable in the dried and also, according to Dr. Low, in the living condition.

* *British Guiana Medical Annual*, 1902.

Of the *Filaria Ozzardi* only a single female and an incomplete male have so far been found. The male of *Filaria Demarquaii* has still to be found. The adult form of the female *Filaria Ozzardi* more closely resembles *Filaria Bancrofti* than *Filaria Demarquaii*, the males of the two differ more markedly, and as in the case in which the *Filaria Ozzardi* were found there were no embryos of *Filaria nocturna* in the blood, there can be no doubt that it was not a specimen of *Filaria Bancrofti*.

Diagrams showing the size and shapes of the cephalic and caudal extremities of these worms with exactly the same magnification are appended. With the exception of the tail of *Filaria Bancrofti* and the head of *Filaria Ozzardi* they are taken from photographs of undistorted specimens. These two, however, are reconstructed from measurements made on the fresh worms. The head of the *Filaria Bancrofti* photographed is in its most extended position and therefore unusually small.

A NEW METHOD OF STAINING THE MALARIAL PARASITES, WITH A DESCRIPTION OF THE STAINING REACTIONS.—C. F. Craig uses the following solutions: Solution A.—A saturated aqueous solution of methyl violet B. This solution should be prepared with distilled water and should be at least three weeks old. Solution B.—A 5 per cent. solution of eosin. The method pursued in staining specimens is as follows: Very thin blood smears are made upon perfectly clean cover-glasses. These smears are hardened in absolute alcohol for from five to ten minutes. They are next carefully dried and stained with solution A for ten seconds; then thoroughly washed in water and stained with solution B for from three to five seconds. The specimens are finally carefully dried and mounted in Canada balsam. As will be seen, the time for staining is very short, which is an improvement over the valuable methods which have heretofore been proposed. The following precautions are to be observed: (1) Both staining fluids should be prepared with the Grubler colours; (2) The methyl violet solution should be at least three weeks old. (3) The smears should be thin and the blood evenly distributed over the cover-glass. In thick smears the colouring is very diffuse and the distinction between the corpuscles and parasites nearly obliterated. (4) The methyl violet solution should not be allowed to act longer than twenty seconds at the most, otherwise the staining is intense. (5) The eosin solution should not be allowed to act for more than five seconds, for the same reason. (6) The specimens should be thoroughly dried before mounting, and thoroughly washed and dried between the applications of the stains. The stain colours the red cells a uniform dark blue, giving to the infected red cells a peculiar and distinctive colour, enabling one to at once pick them out from the others. It differentiates protoplasm, nucleus, and chromatin.—*New York Medical Journal*, September 13th, 1902.

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THE

Journal of Tropical Medicine

DECEMBER 1, 1902.

THE BRITISH MEDICAL ASSOCIATION.

THE COLONIAL COMMITTEE.

THE Committee of the British Medical Association appointed to consider and advise upon Colonial medical matters have important duties to fulfil, if the members of the Committee take up the subject of the Colonial Medical Service seriously. The difficulty of carrying out any real work in connection with the several Committees hitherto appointed by the British Medical Association has been their want of continuity. The Committees were appointed for one year only, and although a Committee might be reappointed, more often than not it was dissolved, and a new set of men took up the work, not usually where the previous Committee left it, but well-nigh *de novo*.

It is impossible to deal with so important a matter as the Colonial Medical Service in three or

four meetings of Committee, for that is usually the number of meetings practically possible during one year. The views of the several members of the Committee are in all probability unknown to each other; and the ideas and objects of men representing the various Colonies may widely differ. The representatives of Crown Colonies have totally different objects in view to those representing the self-governing Colonies; and the Crown Colonies themselves vary so essentially in matters of election, promotion, payment, retiring allowance, &c., that but little advance is possible. Under these circumstances, it was scarcely worth while for a member of Committee to take up any one branch of the many problems that come up before the Committees of the Association, and to hope to get a practical result.

A Committee with a debatable question to deal with, or with one requiring the collection of evidence from out-lying parts of the Empire as obtains in both medical and ethical matters, should be granted a longer life than twelve months. The representatives of the colonies on a Committee may be changed in twelve months, or the Committee may even cease to exist and the medical men resident in distant colonies find that their opinions and conclusions gathered, it may be after careful investigation, are valueless, owing to the member of the Committee representing them being no longer a member of the Committee, or the Committee itself having been dissolved. It would appear to us that the work of many branches of the British Medical Association would be better carried on by Committees elected for three years, or until such time under three years as the work they undertook to do is accomplished. During the period of their existence such Committees should report from time to time to the Council the result of their work, and an annual report should be furnished to lay before the general meeting of the Association.

In some such way as this, interest in colonial medical matters may be kept up; but without such continuity of action in colonial or any other special branch of work, active interest in the Association is impossible.

At the same time it is essential that the

medical officers in the Colonial Service and in the colonies generally, should assist those willing to help them at home by sending in their recommendations and suggestions for the betterment of the Service, or for the closer association of the colonial branches with the governing body. Little advantage is seldom taken in the columns of the medical press of the space willingly granted the more distant members of the profession. Grievances we hear of; but no amount of patch-work can better a service like the Colonial Medical Service. A sop here, or a little more money or leave there, may pacify the claimant for the time being, but something more than that is wanted.

The only scheme before the profession is that of Surgeon-General Evatt's, to which we have often referred, but the medical officers of the Colonial Service have themselves given us no scheme on which to act. Surely from amongst them a "plan of campaign" can be evolved on which the Committee of the British Medical Association, now sitting, can act.

TRYPANOSOMA IN THE BLOOD OF MAN.

WE publish a letter sent to the *British Medical Journal* and the *Lancet*, and appearing in their issues of November 22nd, concerning the notice which we published in the *JOURNAL OF TROPICAL MEDICINE* on November 1st, on "A Case of Trypanosoma in a European." We welcome criticism at all times, and we are obliged to the distinguished men who signed the letter for drawing our attention to what appears to be a subject of contention. We would have preferred had the letter criticising the article in question been sent direct to us, and we should have published it with pleasure. We do so now, however, and we also publish various other letters on the matter which we hope will definitely settle the subject of the discovery of the trypanosoma in man. In the *JOURNAL OF TROPICAL MEDICINE* for September 1st, 1902, page 270, we noticed Dr. Dutton's paper on trypanosoma read at the meeting of the British Medical Association in Manchester. We regret that at the time we did not notice that Dr. Dutton's paper referred to a patient of Dr. Forde's, and that we were publishing in the same issue the original account of this case by Dr. Forde himself.

Dr. Dutton proposes to name the trypanosoma of man the *Trypanosoma gambiense*; but as this parasite was discovered several years ago by Nèpveu in Algeria, and as a name indicating locality is in time likely to prove inadequate, we would suggest that the parasite be named after its discoverer, *Trypanosoma Nèpveui*.

Letter which appeared in the *British Medical Journal* and the *Lancet* of November 22nd, 1902.

NOTE ON THE DISCOVERY OF THE HUMAN TRYPANOSOME.

SIRS,—We have recently seen in the medical press several very inaccurate accounts regarding the authorship of the important new discovery of trypanosomes in human blood, and of the disease caused by them. For instance, the *JOURNAL OF TROPICAL MEDICINE* for November 1st, in giving an anonymous description, supported by an editorial, of a case just observed by Drs. Daniels and Manson, attributes the original discovery to Dr. R. M. Forde. It does not mention even the name of Dr. J. Everett Dutton. Dr. Dutton is an old student and assistant in this laboratory, and is now away on the West African Coast, and we are of opinion that he has a claim to be considered in the matter of this discovery. Another periodical, the *Hospital*, for November 8th, p. 1902, while also omitting Dr. Dutton's name, states that the discovery was made "within the last few days" by the London School of Tropical Medicine. We believe that such statements are calculated to distort the history of the discovery, and should therefore like to have an opportunity for correcting them promptly in your pages.

The facts regarding the history of the discovery—which was made nearly a year ago—have already been publicly and adequately stated both by Dr. Forde* and by Dr. Dutton.† Dr. Forde, Colonial Surgeon, British Gambia, tells us that the case in which the parasites were first observed came under his notice in May, 1901; that he found in the blood "small worm-like, extremely active bodies which I prematurely pronounced a species of filaria," although this conclusion "became doubtful after repeated observations of the parasite"; and that he showed the case in December, 1901, to Dr. J. Everett Dutton, then upon a mission of the Liverpool School of Tropical Medicine to Gambia, and that Dutton "at once recognised" the parasite "as a species of trypanosoma." Dr. Dutton's two papers corroborate these statements of Dr. Forde. After the recognition of the new organism, Dr. Forde gave the first records of the case to Dr. Dutton. Dr. Dutton it was, as Dr. Forde says, who recognised that the fever was of a peculiar undulant type; Dr. Dutton it was who positively excluded malaria as the cause of the symptoms; it was he who saw that those symptoms roughly resemble those of tse-tse fly disease and surra; it is he who has published accurate and able descriptions, drawings and charts of the parasites and of the case; and it is he who is now, with Dr. Todd, investigating the subject in West Africa for the Liverpool School of Tropical Medicine.

Dr. Forde is undoubtedly deserving of great credit for his part in the matter, and we think his name should be associated with the discovery. But until Dr. Dutton was called in, he published no account of the case and did not recognise the nature of the parasite, nor the peculiarity of the symptoms. In order to make a discovery it is not sufficient merely to see an object; it is necessary also to recognise the nature of the object seen, and to publish accurate and adequate descriptions of it. For example, Virchow and others long ago saw the parasites of malaria without recognising their parasitic nature; but it is to Laveran, who did recognise their nature, that science gives the credit for the discovery of them. It is certain that Dr. Dutton was the first clearly to observe and to signal the existence of trypanosomes in human blood, and the first to give accurate descriptions of the new organism; and it is to him that science will give the principal credit for the new observation.

* Forde, *JOURNAL OF TROPICAL MEDICINE*, September 1st, 1902.

† Dutton, *Thompson-Yates Laboratory Reports*, vol. iv., part 2, May, 1902; and *British Medical Journal*, September 20th, 1902, p. 881.

It seems to us particularly unfortunate that the *JOURNAL OF TROPICAL MEDICINE* should have so ostentatiously omitted the name of Dr. Dutton at the moment when it was engaged in giving great prominence to a case of Drs. Manson and Daniels, which, after all, would probably have escaped notice but for the previous work of Dr. Dutton. We may mention also—and this is another point which the *JOURNAL OF TROPICAL MEDICINE* appears to have forgotten—that before his departure for Africa Dr. Dutton gave at this laboratory a detailed demonstration both of the parasite and the clinical features of the case to Drs. Manson and Daniels, and to one of the editors of the periodical referred to. The omission, then, appears to be due rather to want of memory than to want of knowledge. The *JOURNAL OF TROPICAL MEDICINE* also states that while the first case (namely, that of Dutton and Forde) was regarded only as a "curiosity," the "discovery of a second case" (namely, that of Daniels and Manson) "opens up a new field for investigation and elucidation," and so on. This view of the relative importance of an original discovery and of a mere confirmation of that discovery is somewhat novel. But the case of Drs. Manson and Daniels is not the second case at all. The second case—also discovered by Dr. Dutton—was that of a child in British Gambia.

It is unnecessary after what has been said to deal with the statement made in the *Hospital*. It affords, however, an instance of the curiously rapid manner in which such errors are often propagated in the press.

We should note that Barron and Nepveu have also claimed to have found flagellates in human blood; but, as will be seen from their writings, their descriptions are so inadequate as to fail to convince us of the accuracy or even the nature of their observations.

We are, &c.,

RUBERT BOYCE, M.B., F.R.S.,

RONALD ROSS, F.R.C.S., F.R.S., C.B.,

CH. S. SHERRINGTON, M.D., F.R.S.

Thompson-Yates Laboratories, University College,
Liverpool, November 18th.

From the "*British Medical Journal*," November 29th, 1902.

THE DISCOVERY OF THE HUMAN TRYPANOSOMA.

SIRS,—With reference to the letter on the above subject, published in the *British Medical Journal* of November 22nd, from which it would appear, among other things, that this discovery was only made less than twelve months ago, that in my case (the first) malaria was not excluded by me, and finally that I did not recognise the peculiarity of the symptoms, &c., in fact that I simply glanced at the parasite and thought nothing more about it, I wish to make the following statements:—

(1) This parasite was first seen by me in the middle of May, 1901, that is, three or four days after the patient came under my observation. This makes its discovery—if an ordinary medical man be permitted to use the word—to date from eighteen months ago, and not from less than a year.

(2) Malaria was absolutely excluded by me at the time (May, 1901), in consequence of which I made a complete change in my treatment of the case.

(3) I fully recognised the peculiar symptoms of the malady, and was convinced in my own mind that they were associated with the presence of the parasite I found. This fact I mentioned to a colleague at the time, and also to Dr. Dutton on his arrival at Bathurst, before the patient returned from sick leave.

(4) The original temperature chart, the first recorded of the case, taken by me, shows the peculiarity of the fever.

(5) I was quite aware at the time (May, 1901) that I had met with a new disease in man, and fully intended bringing it to the notice of experts in England, but pressure of general work prevented my doing so as early as I wished.

Had the patient informed me as requested, that he had presented himself for treatment at the Liverpool School of Tropical Medicine, I would certainly have communicated to the experts of that institution an account of the case and what I had seen in the blood.

(6) The general condition of the patient, the persistency and novelty of the symptoms, together with the unusual appearance, activity, &c., of the parasite found in his blood, made an indelible impression on my mind at the time.

The extraordinary point about this case is that although the patient was a month or longer under observation at Liverpool during the period he was on sick leave (invalided by me in May, 1901, returned to Bathurst in the following December) the parasite was never seen by the observers, as, according to the temperature chart recorded it must have been present some time or other.

It is stated in the letter referred to above that the case of Drs. Manson and Daniels would probably have escaped notice but for the previous work of Dr. Dutton, I hope it will not be forgotten that the first case would equally probably have escaped the notice of the latter gentleman but for the previous observations, and conviction, as to the novelty of the morbid conditions found, of yours, &c.,

R. M. FORDE.

Senior Medical Officer,

Worthing, November 22nd. Gambia Colony, W. Africa.

SIRS,—We, the undersigned students of the London School of Tropical Medicine, are wishful to animadvert on the letter of Messrs. Boyce, Ross and Sherrington in the *British Medical Journal* of November 22nd. We feel indignant at the tenour of the letter, which, by implication, asperses Drs. Manson and Daniels with filching the credit due to other men for their discoveries, and we shall prove hereby how unjust it all is. Briefly put, the case is as follows:—

A patient has been resident for some time in the Tropical School Branch of the Seamen's Hospital. Dr. Manson very early, from the patient's symptoms, inferred the presence in the blood of the trypanosoma, and communicated to all of us in the course of his routine visits to the wards his conviction that the parasite would eventually be found. In the laboratory, accordingly, Dr. Daniels made careful daily examinations of the blood, and eventually was successful, and repeatedly so, in his quest.

On the day on which the parasite was first found it fell due that Dr. Manson should lecture in due course. He did so, and began by referring to the case, first clearing the ground by disclaiming all originality on his part. He told us that it was a case, the clinical features of which had been demonstrated to him at Liverpool, which had put him on the track of the cause of the one under dispute. He recounted to us clearly the work which had been done at Liverpool in connection with the parasite, told us how it had been demonstrated in the human blood in more than one case by more than one observer, and above all, did full justice to the Liverpool School.

Dr. Daniels, in the laboratory, accorded the same full acknowledgment to the Liverpool School.

You will see, therefore, that in giving all this information to us who will shortly be carrying the facts to all parts of the Empire, these two gentlemen did anything rather than try to filch the credit due to other men for their discoveries. Of course, though some of us can, we cannot all affirm that Mr. Forde and Dr. Dutton were mentioned by name; for men home on leave, snatching information in a hurry, are more concerned with facts than with names, and are very prone to cast aside the latter as useless lumber—this with the fullest respect for scientific pioneers. Could Drs. Manson and Daniels have done more?

All the above notwithstanding, we are all proud of the present case; for we believe that the utilising of the facts gleaned from the Liverpool case by Dr. Manson, in so confidently postulating the parasite in the present case, from

the clinical facts alone, is quite in keeping with his reputation.

"*Tantane animis cœlestibus iræ?*" which may be freely interpreted: Don Quixote was not the only free companion who tilted at windmills.

We are particularly sorry to see appended to the letter the name of Major Ronald Ross, for hitherto, in our estimation, he and Dr. Manson have always stood together in our list of medical worthies; and we trust that we shall never again see anything in print which may lead to the suspicion of discord between the two.

We are, &c.,

C. J. BAKER,

G. HOOD,

E. LANGLEY HUNT,

G. IVANHOE LECESNE,

RAOUL F. DE BOISSIÈRE,

AUBREY H. DAVIES,

EMMELINE DA CUNA,

J. LUNN,

R. A. BELILIOS,

WILLIAM FLETCHER,

M. E. O'DEA,

TAYLOR HANCOCK,

GEOFFREY HUNGERFORD,

M. CAMERON BLAIR.

London School of Tropical Medicine,

Royal Albert Dock, E., November 22nd.

To the Editors of the JOURNAL OF TROPICAL MEDICINE.

SIRS,—Being responsible for the reference to trypanosoma which appeared in your issue of November 1st, I feel that it is my duty to answer the very formal protest advanced against it in the *Lancet* of 22nd inst., by Prof. Rubert Boyce, Major Ronald Ross and Prof. Sherrington. These gentlemen's protest has surprised me in the extreme, and I am bound to say that, even now, I am unable to grasp the real motive which prompted their letter. Anyhow, I will suppose that they considered my communication an encroachment on their rights of priority similar to that which was unfortunately made soon after Dr. Low's demonstration at the London School of Tropical Medicine, that the larval *Filaria bancrofti*, having reached a certain stage of development in the thoracic muscles of *Culex fatigans*, migrates to the insect's trophi to be subsequently inoculated into a fresh human host.

Had my communication been so devised, it would not yet have been a parallel case, because I sent it to a well-informed medical journal, whilst their notification appeared in the first instance in the lay press.

Anyone who will read my communication with an unbiassed mind, will, I am sure, be unable to see adequate reason for their protest. I did not attribute the discovery of the presence of trypanosoma in man either to Dr. Manson, or Dr. Daniels, I simply stated that Dr. Manson, having had the opportunity of examining Forde's patient in Liverpool (already described in this Journal a few weeks previously), was able to diagnose a second case which was subsequently studied at the London School of Tropical Medicine, and that Dr. Daniels had found, and of course recognised, the trypanosoma just as Dr. Dutton had recognised the nature of the parasite found by Dr. Forde in his case.

Further, I added that the presence of trypanosomes in a second case presenting the same striking and grave symptoms observed in Dr. Forde's patient, showed that the presence of trypanosomes could no longer be regarded as a mere curiosity like many erratic parasites occasionally found in man, but must be looked upon henceforth, in certain regions, as an important pathological factor.

I did not sign my hasty communication, because its object was not that of advertising my name, and I stated that an accurate account of the case would be published very shortly by those who were investigating it.

I did not mention Dr. Dutton, because it did not occur to me that he had anything whatever to do with the case I was reporting, and even now I utterly fail to see why I should have mentioned his name. In other publications on filaria, &c., I have, at opportune times, mentioned Dr. Dutton's

excellent work. In this instance, I was not writing in praise of the Liverpool School; that has been done lately to a large extent. I was merely mentioning an interesting case of trypanosoma infection.

Having explained the reason of my anonymous communication, I must now myself protest against the very gratuitous accusation brought by Drs. Boyce, Ross and Sherrington, when they say:—

"We believe that such statements are calculated to distort the history of the discovery and should therefore like to have an opportunity for correcting them promptly in your pages."

These gentlemen virtually accuse me of having attributed the discovery of the presence of trypanosoma in man to Drs. Manson and Daniels, a statement which I did not make, nor ever dreamt of making. But what is still more surprising is that while they accuse me of distorting the history of this discovery they ascribe the honour of having made it to "an old student and assistant" of their laboratory!

I am sorry that Major Ross, who has so strongly resented Prof. Grassi's encroachments on his work, should now set aside information of which he is evidently aware, even for the purpose of claiming for his own school the discovery of trypanosoma in man.

Major Ross and his colleagues, after having stated that "Dr. Dutton was the first clearly to observe and to signal the existence of trypanosomes in human blood, and the first to give accurate descriptions of the new organism," add, without mentioning dates, and in the most unfair manner: "Barron and Nepveu have also claimed to have found flagellates in human blood, but as will be seen from their writings, *their descriptions are so inadequate as to fail to convince us of the accuracy or even the nature of their observations*" [the italics are mine].

To disprove these erroneous statements I can do no better than quote verbatim Dr. G. Nepveu's very clear account of his discovery of trypanosomes in man, which, be it noted, he made between 1890 and 1898, and published in the *Comptes rendus des séances de la société de Biologie*, on December 24th, 1898, many years before Dr. Dutton's publication. Dr. Nepveu writes as follows:—

"Trypanosomes have been observed only in the blood of animals; in India they have been found in the blood of rats (Lewis), horses (surra epidemics), dogs and domesticated elephants. In Africa they have been discovered in the disease caused by the tse-tse fly, and in Europe in the blood of rats, rabbits, birds and frogs."

"No one seems to have found them so far in man; however, Laveran (1) states that Barron seems to have found certain flagellated protozoa of undetermined genus in the blood of an anæmic woman."

"In 1890, in consequence of researches made in Algeria on the malaria parasite, I have found, in the blood of a patient, besides *Laverania*, a flagellate which seemed rather common, because I could count about three in each preparation of 18 square millimetres. At about that time (See Nepveu, *Etudes sur les Parasites du sang chez les paludiques*, 21, 1891, in *Bulletins et Memoires de la Société de Biologie*), I already published some of the drawings I had so far collected. I then hoped I might complete my first observations by a more detailed study, but since that time only very rarely have I been able to find that parasite. I have therefore decided to publish the following facts in the hope of drawing the attention of those naturalists and physicians who will have the opportunity of completing these researches."

"This trypanosoma presents all the characters of the genus: general shape a homogenous colourless membrane, one border of which is thinner, hyaline, and presenting characteristic undulating movements. This membrane bears a nucleus and a fine flagellum placed anteriorly; the undulations of the latter follow in rapid succession."

"Thus it presents itself in the patient Khill (quotidian fever), in Cabane (pernicious comatose fever); in a third

patient, Ginestet, I have found certain organisms which I thought I might compare to those described under the name of Trypanomones, a form which is probably only an evolution stage of the trypanosome. In this patient the organisms were provided with two flagella at one of their extremities. Labbé has described such trypanomones."

"On over two hundred patients, mostly malarial, of which I have examined the blood, I have only found these various forms in six. Three of these were suffering from quotidian fever (Khill, Langevelde, Bichielli), one a double tertian (Hendrick), two the pernicious comatose fever (Cabane and Ginestet); the seventh observation was on Dr. X., who was apparently in good health."

"In none of these patients have I been able to observe any symptoms characteristic of this special parasitic invasion. They were almost every one of them suffering from the effects of *Laverania* which prevailed on all and everywhere in its various forms. This seems therefore purely and simply a coincidence which has seemed to me worthy of notice."

"In conclusion, the trypanosome must be classed amongst the parasites of human blood. I am unable at present to give with the necessary precision a more complete description of this variety, I therefore abstain from giving it a special name. It is better to establish in the first place the analogies and differences which I perceive between this parasite of man and the congeneric parasites in animals, and also to complete the observations on its morphology and life history."

The above quotation speaks for itself, and I will only add that there can be no question about Dr. Nepveu's *bona fides*. The two-flagellated organism he mentions in his paper is so peculiar a feature that it is not likely to be imagined. That it is a feature of the trypanosoma of man I can attest, because Dr. Daniels found it in the blood of our patient.

It is to be regretted that the representatives of the Liverpool School of Tropical Medicine, in their praiseworthy endeavour to make a new discovery, should have overlooked previous work.

Your obedient servant,

LOUIS W. SAMBON, M.D. (Naples).

Lecturer to the London School of Tropical Medicine.

British Medical Association.

NOTE ON A TRYPANOSOMA OCCURRING IN THE BLOOD OF MAN

By J. EVERETT DUTTON, M.B. Vict.

Walter Myers Fellow, Liverpool School of Tropical Medicine.

WHILE staying at Bathurst, Gambia, last year, for the purpose of investigating the sources and distribution of mosquitoes in the town on behalf of the Liverpool School of Tropical Medicine, I was asked by the Colonial Surgeon, Dr. R. M. Forde, to examine the blood of one of the Government officials who had just returned from England, having been invalided home six months previously, after an attack of "fever" which had proved quite resistant to treatment with quinine. In the blood of this man I found a flagellated protozoon evidently belonging to the genus *Trypanosoma*. The clinical history of the case is as follows:—

Mr. K. is an Englishman, aged 42, who has been for the past seven years in Government employ, with intervals of leave, as master of the Government boat plying weekly up the River Gambia. His illness dates

back to May, 1901, when he broke down after the especially heavy duty occasioned by a punitive expedition up the river. Previous to that time he had enjoyed good health, except for very occasional slight attacks of malarial fever which were amenable to quinine. On May 10th, 1901, he was admitted to the hospital at Bathurst with fever, under the care of Dr. Forde, who very kindly gave me a copy of the temperature chart (Chart I.)* Fresh preparations of the patient's blood were examined at that time; no malaria parasites were seen, but Dr. Forde informed me he saw many actively moving worm-like bodies, the nature of which he was unable to ascertain. On June 1st, after three weeks in hospital, the patient was invalided home, and on August 12th admitted to the Royal Southern Hospital, Liverpool, under the care of Dr. Macalister, to whom I am indebted for some notes on the case. At this time his chief troubles were general weakness and pain in the left side.

The chief features presented by this case during his stay in the Royal Southern Hospital were as follows:—

The temperature, which on admission was sub-normal, was marked throughout by slight rises above normal, and three more important elevations to 101° to 102°, lasting a few hours and rapidly falling to normal. The pulse—on admission 120—fell in the evening to 92; the pulse-rate continued always fairly rapid, at about 100 per minute, and increased with the slightest exertion.

The respiratory rate was also similarly increased, being 32 on admission, falling later on to 20 per minute, and afterwards keeping about 25.

The liver showed slight enlargement, and the spleen, described as being normal on admission, could afterwards be felt below the ribs, and was then very painful; there was throughout considerable tenderness over the splenic area.

Beyond some slight dyspnoea on exertion the respiratory system was apparently normal. The heart sounds were weak and distant. Patient often complained of weakness in the legs.

The blood was examined for malaria parasites on two different occasions, but none were found.

Patient left hospital improved, the pain over the spleen having disappeared. After recuperating, he returned to Bathurst in the early part of December, 1901. During the voyage out he had a severe illness which was diagnosed by the ship's surgeon as an atypical attack of pneumonia. On landing at Bathurst it was generally remarked that he was very much thinner and easily fatigued. He was placed on the sick list, but his condition did not necessitate admission into hospital. Dr. Forde and myself made an examination of the patient on December 18th. We found his temperature 100.4°, pulse 96, respiration 34. Weakness of the legs and a little breathlessness on exertion were the chief troubles complained of. There was also a little loss of appetite and sleeplessness at times. On December 16th he had had a slight bleeding from the nose.

The general facial aspect, which has been remarked

upon by his friends, attracted our attention. The face was distinctly puffy and flushed, the eyes appeared sunken, and the conjunctivæ watery; there was a distinct fulness of the lower eyelids, which were found to pit on pressure. On examining the body generally the skin was distinctly congested in places, especially the chest and thighs; there were seen irregularly scattered, purplish coloured areas, due to a localised congestion of the superficial capillaries; after pressure with the finger in these regions the purplish colour only very slowly returned.

Around the ankles there was some œdema, the skin in this region pitting on pressure. A systematic examination of the various systems was made, but nothing abnormal was detected with the exception of distinct enlargement of the spleen, which organ could easily be felt below the costal margin; dullness measured diagonally 7 inches; a slight bulging was noted over the splenic area. The temperature, pulse, and respirations (Chart II.)† during the time patient was under observation were very similar to the previous records; from the charts it will be seen that the temperature recorded was never very high, as a rule not much above 101°. The course of the temperature was rather irregular, but of a distinctly relapsing type, thus showing a characteristic of the temperature charts of such animals as the ass and mule infected with surra or nagana, which diseases are known to be associated with the presence of a trypanosoma in the blood. Also as seen in these animals, the number of parasites encountered in the blood was found to vary with the temperature—thus, from December 16th to December 18th, when the patient's temperature was raised, parasites were found in the blood, the largest number counted in fresh preparations being fifteen under a $\frac{3}{4}$ -inch square cover-glass. On December 19th the temperature fell below normal, and on that and subsequent days no parasites were detected, but they reappeared about December 27th during a fresh attack of fever.

The patient's condition improved during the week which he spent at the Cape, seven miles from Bathurst, at the mouth of the Gambia River, where there is an excellent Government House facing the sea. He afterwards was allowed to resume his duties, and took the Government launch up river on January 5th, 1902.

The most notable features presented by this case were:—

- (1) Its chronic course.
- (2) The general wasting and weakness.
- (3) The irregular rises of temperature, never very high, and of a relapsing type.
- (4) The local œdemas.
- (5) The congested areas on the skin.
- (6) The enlargement of the spleen.
- (7) Constant increased frequency of pulse and respiration (hurried breathing).

This condition still persists; the general weakness has increased, in consequence of which the patient has been again invalided home, and arrived in England a few days ago.

* For Chart I. see Dr. Forde's article in the JOURNAL OF TROPICAL MEDICINE, September 1st, 1902, page 262.

† For Chart II. see Dr. Forde's article in the JOURNAL OF TROPICAL MEDICINE, September 1st, 1902, page 262.

THE PARASITE OBSERVED IN THE BLOOD.*

Although many slides were made and fresh preparations of the blood examined throughout the time the patient was under observation, no malaria parasites were discovered. The first examination of the blood was made on December 15th. On that day I made three fresh preparations, using a $\frac{3}{4}$ -inch square cover-glass. In these preparations I observed altogether three parasites, presenting all the characteristics of the trypanosomata.

In fresh blood the parasite appears as a very minute worm-like organism, very difficult to see with a magnification of 300 diameters; especially in this case, when only a few are present in a preparation, and the parasite is entangled amongst a clump of red corpuscles; it glides along fairly rapidly in among the red cells, imparting very little movement in them. When the movements have slowed down one end of the organism is seen to be drawn out into a whip-like process—the flagellum; the other end is bluntly conical; attached along one side of the body is a transparent flange-like process—the undulating membrane; the body itself is short and thick, and its substance granular. There is a highly refractile spot situated near the posterior end (vacuole).

The parasite usually is seen progressing with the flagellum, which represents the anterior end, in front, but at times when an obstruction is insurmountable it shoots backwards for a short distance with the blunted end (posterior) forward. Progression is brought about by wave-like motions started in the flagellum and communicated along the undulating membrane, also by contractions of the body protoplasm. The parasite in rapid motion moves in a screw-like manner, its body rotating around the longitudinal axis, so that the undulating membrane appears as if it were spirally arranged around the organism.

On one occasion I observed the process of phagocytosis take place on a slide one hour after the blood was drawn; a mononuclear leucocyte had partially englobed the trypanosome, only the flagellum and a small portion of the anterior part of the body remaining free.

In fresh preparations, ringed with vaseline, the parasites appear to die in a few hours after the blood is drawn (one observation three hours). In such preparations, left over night, I was never able to find the trypanosoma again in the morning. Atmospheric temperature varied from 90° in the day to 65° during the night. I was unable to obtain an exact measurement of the parasite in the fresh state.

Blood films were stained by a modification of the method of Romanowsky for chromatic staining which had been devised by Dr. MacConkey, to whom I am indebted for the formula. (Dr. MacConkey, I understand, will describe his method at an early date.)

The length of the parasite, in stained preparations, including the flagellum, varied from 18 μ to 25 μ . In preparations which were taken on December 16th (first observation) the parasites appeared somewhat longer than those taken when they appeared in the blood again on December 27th; the majority of

specimens measured 22 μ ; the width was 2 μ to 2.8 μ . This width, when compared to the other trypanosoma, is distinctly greater in proportion to the length.

The flagellum stains a light crimson, and can be traced from the anterior end of the organism along the outer margin of the undulating membrane, and appears to end near the refractile spot seen in fresh preparations; it sets in small curves along the body, and there is always present a dip opposite the nucleus. The free part of the flagellum is about one-third that of the total length, but it is difficult to say where the anterior part of the body ends and the flagellum begins. One can always see a narrow streak of protoplasm, staining blue, for some distance beneath the free part of the flagellum.

The posterior end of the organism ends abruptly and is roughly conical, in most specimens with the point of the cone cut away on the side remote from the undulating membrane.

The undulating membrane is a narrow unstained band, somewhat wrinkled, attached along one side of the animal; in stained preparations it sometimes takes on a faint pink colour.

The nucleus (the macronucleus of Plimmer and Bradford) is situated a little anterior to the middle of the body, in some specimens occupying the whole width of the animal; it is oval in shape, and stains dark crimson, due to an aggregation of chromatin granules.

Generally about 2.5 μ from the posterior end is a dark purple spot, well marked, showing no definite structure; this is the centrosome (Laveran and Mesnil), or micronucleus (Plimmer and Bradford). The flagellum is intimately connected with the micronucleus. Anterior to it there is a large clear spot (vacuole) which does not stain; the vacuole in all the specimens is well marked.

The protoplasm does not stain evenly; it takes on a basophile reaction, and in it in some specimens are fine blue-stained granules situated chiefly around and in front of the macronucleus. The organisms "set" in a characteristic manner on a slide, the body is generally bent at an angle opposite the nucleus. I have observed this in most of the stained preparations; whether it is a distinguishing feature or not is difficult to decide, but it is curious to note in film preparations the body of *T. lewisi* does not bend but sets in a crescentic manner; in the case of *T. brucei* the body makes three or four curves.

I have not observed dividing forms in any of the slides made. The blood in this case showed no very marked anæmia, the red corpuscles numbered 3,850,000 and white corpuscles 12,000 per c.mm.

A differential count of white corpuscles was made on several occasions when the parasites were present, and when few or none could be detected in the blood. On all occasions the counts showed an increase of lymphocytes at the expense of the polynuclear leucocytes, the relation being generally about 50 per cent. of the latter to 40 per cent. of the former.

I have examined a series of 115 blood films obtained from native children (aged 1 to 15), for the purpose of estimating the prevalence of endemic malaria in the Gambia.

In one preparation of blood taken from a child,

* See plate illustrating Dr. Forde's article in the JOURNAL OF TROPICAL MEDICINE, September 1st, 1902.

aged 3 I found trypanosomata present. In the smear three parasites were counted, presenting identical characteristics—size, shape, staining reaction, and position taken up on the slide—to the parasite described occurring in the blood of the European; associated with the trypanosomata were a few ring forms of malaria parasites.

The child was one of a batch of fifty examined at a native village seven miles from Bathurst, near the mouth of the River Gambia; these children were to all appearances healthy.

Up to the present time four previously well-known diseases occurring in lower animals in various parts of the world have been shown to be associated with the presence of trypanosomes in the blood. These are:—

(1) Surra, the organism of which was discovered by Dr. G. Evans in 1880, in horses and other animals in India, North-West Provinces.

(2) Nagana, in Central Africa and in other parts, attacking horses, cattle, and other animals. Bruce discovered the parasite in 1894.

(3) Mal de Caderas, in Central South America and Brazil: the disease is very similar to surra and nagana, and is produced by a trypanosoma probably identical with that of *T. brucei*.

(4) Dourine, or *Maladie du Coût*, occurs in Algeria, South France, Spain, and Turkey, the pathological agent of which is the *T. equiperdum* (Doflein), *T. rougeti* (Laveran).

In February of this year Lieutenant-Colonel Bruce reported a discovery by Dr. Theiler of a new trypanosoma which is pathogenic for cattle in the Transvaal. Horses, dogs, goats, rabbits, and guinea-pigs appear immune.

Comparing the symptoms which have been described in animals suffering from these diseases with those which I have observed in the patient during the short time he has been under observation, they appear to have many points in common, namely:—

(1) The chronic course of the disease. The illness in my case has already lasted fifteen months. In cattle, sheep and goats nagana runs a chronic course of some six to eight or more months. In cases of surra in cattle and of dourine in horses, the respective disease may last a similar length of time.

(2) In all animals infected with a pathogenic trypanosome marked cachetic symptoms are characteristic. The present case during the fifteen months has lost 26 lbs.

(3) The gradually increasing weakness, especially in the legs and arms, in this case correspond to that most striking symptom which gives to Mal de Caderas its name; loss of power and paresis progressing to paralysis are present also in surra and nagana.

(4) Localised and fugitive α -demas occur in all the animal affections. I have already remarked their occurrence in the present case.

(5) Some pathological condition of the small vessels and capillaries giving rise to congested patches, petechie, and hemorrhages are often associated with other symptoms in animals. The peculiar condition of the skin and the bleeding from the nose I have already referred to in the description of the symptoms of this patient.

(6) A peculiar tendency to a relapsing type of fever is shown by the temperature charts of many infected animals, especially those in which the disease assumes a more chronic course, and also has been noted in the human affection. As far as my observations go there appears to be also a relation between the presence of the parasites in the blood and the elevation of temperature similar to that which has been described in nagana and surra.

In contrasting the parasites with similar parasites in animals it approaches most nearly in its morphology the *T. brucei*. It is the smallest of all described mammalian trypanosomata; its average length is $22\ \mu$ including the flagellum; its breadth is greater in proportion to its length than in other parasites. The posterior part as measured from the micronucleus to extreme tip is short and characteristic for this parasite.

The micronucleus and its associated vacuole are always large and well marked. The "set" in fixed specimens differs from that of other species as has already been pointed out.

Dr. Laveran, who has very kindly examined some blood films taken from the patient, informed me that if the morphological characters are alone considered he would regard my specimen as a new species, it differs from *T. brucei* in the length of the flagellum and by the small number of chromatin granules in the protoplasm.

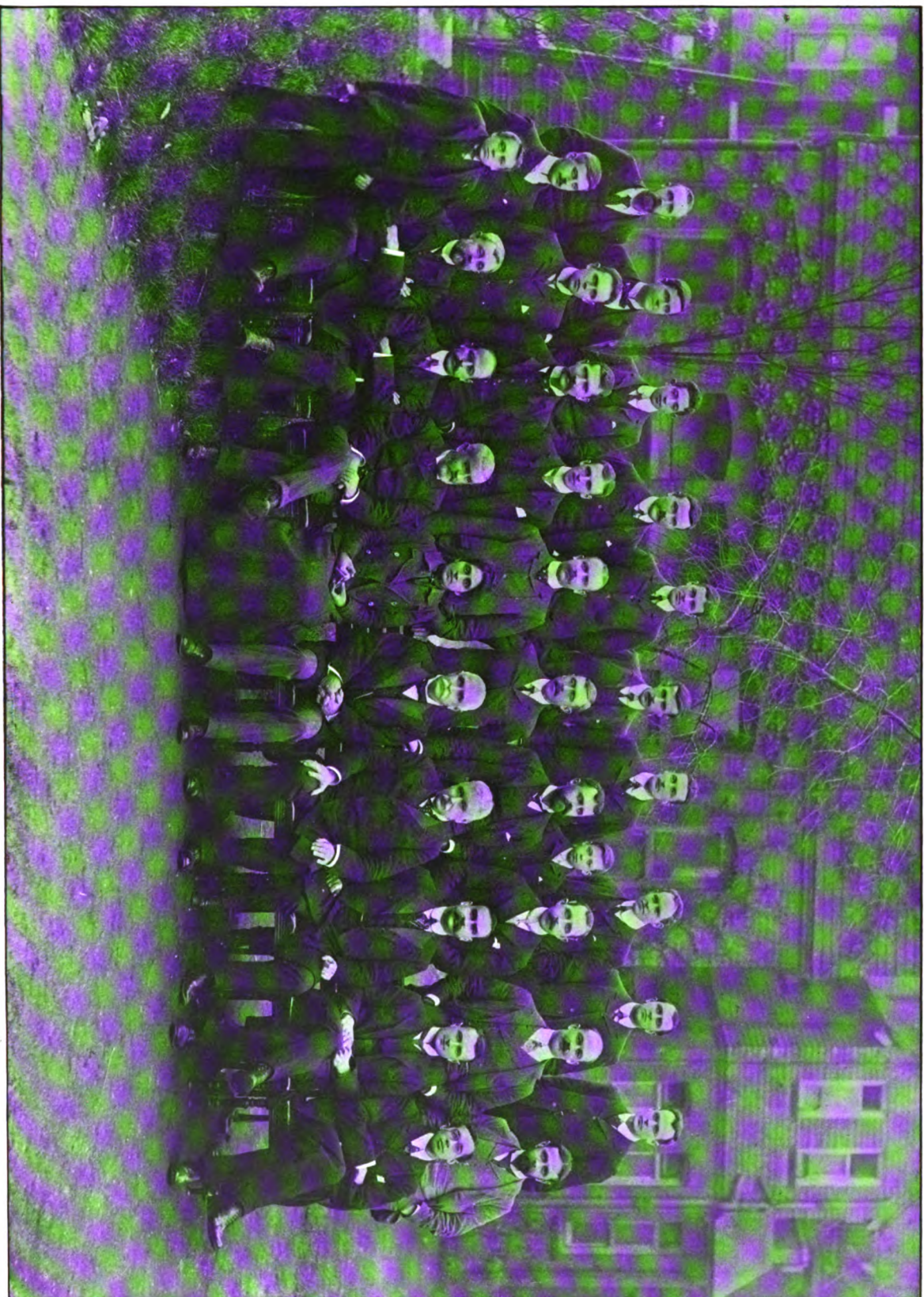
Having as yet not had the opportunity of transferring the parasite in the blood from man to other animals as has been so completely done in nagana by Bruce in Africa; Kanthack, Durham and Blandford, and Plimmer and Bradford in England; Laveran and Mesnil in France; and to a less extent in surra by Evans, Steel, Lingard, Vandyke Carter; and in dourine by Rouget, Nocard, and others, I am quite unable to contrast the pathogenicity and the morphological appearance of the human parasite in lower animals with the other species. It is to be remembered that no case has ever been recorded in man in the districts in which animal infection is so common, although man is exposed to the same risk of infection; for instance, the tse-tse fly (*Glossina morsitans*, Westwood), which was proved by Bruce to carry the infection of nagana from animal to animal, bites travellers, natives and others, as well as animals.

The consideration of these facts and the discovery of a parasite—evidently of the genus trypanosoma—in the blood of a patient presenting symptoms markedly similar in very many points to those of the two or more diseases of lower animals which have been definitely proved to be caused by the presence of different species of the genus trypanosoma forces one to the conclusion that the parasite found in this patient is a new species, and is also the cause of the disease from which the patient is suffering. I would therefore suggest that the name of *Trypanosoma gambiense* be given to this trypanosoma.

In conclusion, I wish to take this opportunity of thanking Dr. Forde, who kindly allowed me to investigate the case at Bathurst, Dr. Laveran, Lt.-Col. Bruce, Dr. Plimmer, Dr. Rose Bradford, and my colleagues at the Liverpool School of Tropical Medicine for the great interest they have shown, and for

STUDENTS AND SOME OF THE STAFF OF THE LONDON SCHOOL OF TROPICAL MEDICINE.

10th Session, October-December, 1902.



R. T. Herdman, S. P. Pearl, G. Levesne, M. E. O'Dea, J. Linn, D. Steel, W. Fletcher, G. Duncan Whyte, G. B. Warren (Lab. Assistant), M. Sandeman,
A. H. Davies, A. Balfour, M. C. Blair, W. S. Milne, J. T. Hancock, C. J. Baker, T. Hood, Charles (Lab. Boy), W. J. J. Stewart, E. Langley Hunt, C. W. Daniels (Med. Superintendent),
Robert (2nd Lab. Assistant), D. Christie, R. A. Bellios, O. Galecy, Miss E. da Cunha, Sir Francis Lovell (Dean), Dr. P. Manson (Lecturer), Dr. L. W. Sambon (Lecturer),
G. Hingston, R. F. De Boissiere.
Other students are not in the photograph.

many suggestions. I am indebted also to Dr. Annett, who has kindly promised to undertake the investigation of the case on my departure to West Africa.

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Translation.

TSE-TSE DISEASE IN TOGO (WEST AFRICA).

By Dr. HANS ZIEMANN, Naval Staff-Surgeon.

(Translated from the German by P. Falcke.)

AT the commencement of June, 1900, my Governmental term of medical practice being concluded, I was, on my return journey, invited by the former Official Medical Officer of Little Popo, in Upper Guinea, Staff-Surg. Bludau, to avail myself of the resources of the Nachtigal Hospital, and devote myself to the study of the diseases of Togo. I accepted this invitation gratefully for a period of a month, so Dr. Bludau was in a position to follow the work with me. I was likewise considerably assisted by the officials and merchants of Little Popo. Although my attention was principally directed to the investigation of the etiological conditions of malaria amongst the white population, and also the diseases of the natives, some attention was also directed to veterinary pathology. In this connection I succeeded in confirming the existence of Tse-tse disease in Togo, and only want of time prevented me from also approaching the question of the occurrence of Texas fever in Togo, as the ticks (*Irodidae*) which transmit the disease were observed by me in that

country. The possibility, or rather the *great probability*, of the occurrence of that disease in the hinterland of Togo and Cameroon are set forth in a report to the Colonial Department. At any rate, the occurrence of tse-tse in Togo was quite unknown to my attendants until the period of my visit. In Cameroon, however, the suspicion of the existence of tse-tse in Upper Guinea had already gained ground in consequence of the symptoms presented by horses from that district.

I now learned in Little Popo that a horse-disease developed in the hinterland of Togo when healthy horses were taken beyond the seventh degree of latitude. The district of Misa Heights is supposed to be afflicted very severely. One particularly characteristic sign was that in this disease a thick cord appears beneath the skin, which extended from the neck to the scrotum, from which when incised a fairly clear fluid exuded. The Haussas, the well-known commercial people of the hinterland of Upper Guinea, treated the disease with enemata and purgatives. When the animals were much reduced the Haussas were supposed to feed them forcibly by stuffing balls of millet (Durra) mixed with maize down their throats.

At that time nothing more was known respecting the disease. My search for the tse-tse fly (*Glossina morsitans*) in Little Popo and vicinity was not attended by success; but it must be noted in this connection that being at that time engaged in the investigation of malaria, I seldom went beyond the lake. The natives also professed never to have even seen a fly answering to the description in Little Popo. Chance, however, provided the necessary material; a communication on this subject was laid before the Paris Congress, 1900,* but did not appear to arouse further interest. In 1901 and 1902, Dr. Schilling† published some interesting researches on surra (synonym for tse-tse disease) in Togo. The author being permanently on the spot had plentiful material at his disposal, and his investigations are a valuable supplement to my discoveries, which, however, differ partly from his. Dr. Schilling's last essays also contain a hopeful outlook for the prophylaxis of this pestilence which is so fatal to cattle.

I cannot discover from Schilling's words if he had any knowledge of my researches, likewise conducted in the Nachtigal Hospital eleven months previously.

Here follow the notes made in his time:—

CASE 1. June 27, 1900.—Terrier bitch, 4 years old, born in Europe, has been in Little Popo for three years. Has never been ill previously; was brought to me by her master. The terrier had littered three months previously; has never been into the interior. One of her litter had died two

* Zweiter Bericht über Malaria und Moskitos an der afrikanischen Westküste. Deutsch. med. Wochenschr., 1900. Also Bericht über die Sitzungen im Institut Pasteur in den Verhandlungen des Congresses zu Paris.

† Dr. Schilling: (a) Bericht über die Surra-Krankheit der Pferde. Centralbl. f. Bact. u. Th. Bd. xxx., 1901, No. 15. (b) Bericht über die Surra Krankheit der Pferde und Rinder im Schutzgebiet Togo. Centralbl. f. Bact. u. Th., Bd. xxxi., 1902, No. 10.

months previously with the same symptoms as are described below. The remaining two puppies of the same litter are healthy. Until this morning the animal, which is always narrowly watched by her master, was apparently well. This morning the terrier was seized with vomiting and diarrhoea. The food, as far as could be vouched for, had always been the same. The vomit and stool were asserted to have looked greenish. The temperature at 3 p.m., taken per rectum, was 38.2° C. The animal appears to be seriously ill. Respiration 44 per minute, heart beat 140. The bitch cannot stand, and when she tries to she falls on her side after rocking to and fro. No erythema of the skin; no oedema. Several tonic and clonic cramps of the head and limbs, which are, however, of short duration, never longer than one minute. Some mucus, which unfortunately was not examined, flowed from the nose and mouth. The pupils react to light. Reflex of the cornea maintained. There is frequent anguished howls, but only in the intervals between the convulsions. The examination of the blood exhibits a remarkable number of parasites, which in their morphological appearance may be regarded as tse-tse parasites (description below). There is a slight degree of leucocytosis. A few red blood corpuscles exhibit metachromatic colouring. The following animals were inoculated subcutaneously, each with $\frac{1}{2}$ ccm. defibrinated blood with $\frac{1}{2}$ ccm. 0.6 per cent. sterilised solution: (1) One duck; (2) one cock; (3) one pigeon (tame); (4) one kid, a short-legged variety of the Togo goat; (5) one small black sucking-pig from the farmyard of a Ewe negro.

The blood examination of these animals exhibited no deviation from the normal excepting that of the pigeon, which contained a few halteridium. [Some blood of the terrier placed on agar-agar tubes.] The condition became worse at 6 p.m., the temperature was 39.8° C. The tonic and clonic convulsions became more frequent and unconsciousness supervened, the terrier lying on her side with eyes wide open and fixed. Death at 3 a.m. Obduction seven hours later. [The histological examination is not yet concluded.] Marked rigor mortis. The terrier is somewhat lean. A little saliva has dribbled from the mouth. No marks of bites on the tongue. No erythema and oedema in the skin. The conjunctivæ are of a dirty yellowish colour, very moist. The lymphatic glands are not enlarged. A few ecchymoses on the pericardium. The subcutaneous cellular tissue of the abdomen somewhat discoloured and is oedematous, infiltrated to a slight degree; the heart is not enlarged but is flabby. Muscles yellowish-brown, parenchyma dim. There are clots in the ventricles and auricles.

The lungs normal. No ecchymoses are observable on the pleura. No exudation into the abdominal cavity. Liver enlarged, reddish-brown in colour, somewhat harder than normal, permeated by a number of whitish-yellow spots from the size of a lentil to a pea; the smaller spots are differentiated sharply from the reddish-brown vicinity and are found to consist of altered hepatic tissue, in which no trypanosomes are present. Portal vessels full of them in parts.

The biliary capillaries are also very full. Kidneys not enlarged, of normal consistency. Capsule easily removable. Cortal substance opaque with striped reddening of the medullary substance. The urine taken from the bladder exhibits traces of albumen.

Slight parenchymatous gastritis. Intestines normal macroscopically. No ecchymoses on the serous membranes. Spleen slightly enlarged, chocolate-coloured, and of deliquescent consistency. No trypanosoma in streak preparations. Unfortunately the mammary glands were not examined.

Brain.—Slight hyperæmia of the cerebral vessels. meninges easily removable; otherwise no abnormalities macroscopically. Bone marrow from femur exhibits no peculiarities, no trypanosoma.

June 29.—Blood examination of the inoculated animals negative; all lively. No growth observable on the agar-agar tubes.

July 2.—Blood examination of the inoculated animals negative.

July 4.—Blood examination of the inoculated animals negative.

July 5.—Halteridium unchanged in the pigeon. No trypanosoma in either the duck, cock, or pig. Growth on agar-agar tubes — 0. The kid exhibits a great number of trypanosoma in the peripheral blood resembling the trypanosoma of the dead terrier. The kid has become somewhat languid. Appetite slightly diminished. No paresis or convulsions. No glandular enlargements; no erythema or oedema. Temperature at 5.30 p.m., 41°.

July 7.—Temperature at 7 a.m. is 39.6°. The kid is fairly lively. Being taken on board ship the blood was not examined.

July 8.—Temperature at 7 a.m. is 39°. The kid is lively and enjoys its food. At first no trypanosoma can be found in the living blood. Two trypanosomes are found in stained preparations after a long search. At 6 p.m. temperature 39.6°.

July 9.—Temperature 39°. Still a large number of trypanosoma in the blood.

July 10.—Temperature 38.4°. Still a large number of trypanosoma in the blood.

July 11.—Temperature 38.9°. Very few trypanosoma.

July 12.—Blood examination gives negative results.

July 13.—Very few trypanosoma. The animal is very lively.

July 14.—Two trypanosoma found in four stained preparations after a long search.

July 15, 16 and 17.—Blood examination negative.

July 19.—One trypanosoma found in preparation after a long search.

July 20 and 21.—Blood examination negative.

The kid was sent to the Institute for Infectious Diseases in Berlin for the study of methods of immunisation, and after repeated blood examinations was pronounced healthy. The periodical variations in the occurrence of the parasites in the blood should be noted, they being most numerous on those days when the temperature was highest. In this respect the infection described above resembles the real surra as observed in India.

CASE 2. June 28, 1900.—Bobbie, terrier, 4 years

of age, the property of Staff-Surgeon Bludau. Born in Europe, has been in Togo for three years, and during the last few months has always been in or near Little Popo. Hitherto has always been healthy.

During the forenoon the dog, usually a very lively animal, exhibits great fatigue and sleepiness, and does not eat. Indication of tonic, slight and rapidly transient, convulsions of the limbs; otherwise nothing abnormal. Temperature, 38.7°. Blood examination quite negative, and also on following day, when the animal has again become lively.

At first it was conjectured that both dogs were suffering from some malaria-like ailment—a sort of Texas fever—as *Ixodidae* were found on another large dog, a St. Bernard, belonging to Dr. Bludau; these, however, could not be identified as unfortunately the preparation was lost.*

As long ago as 1894 I had succeeded in finding blood parasites in a pug on the West African Coast; morphologically these parasites resembled the parasites of tropical fever, and many forms of *Piroplasma bigeminum* are known to be hardly distinguishable from these. This dog had also died from convulsions.† This was long before Marchoux, in Senegal, made his discovery of corresponding blood parasites in the dog, being the first confirmation of malaria-like parasites of dogs in the tropics.

In regard to Case 2, the clinical symptoms and the fact that, as we shall see, tse-tse may occur in a very mild form, lead one to conjecture that Dr. Bludau's terrier was also suffering from the disease.

CASE 3.—Horse belonging to Mr. Keutzler, about 4 years old, has been in Little Popo, Togo, about three years, having been imported from Lagos. On June 27th it was reported that the horse has fallen off during the last two days; appetite not good and does not drink much. On the abdomen, proceeding from the penis towards the front, there is a flat thickening about 15 cm. in length and 3 cm. in breadth. The skin over it is immovable, the glans penis slightly oedematous. Oedema is not perceptible on the legs. Temperature per rectum 38.2°. At first no trypanosoma are observable in the living blood. In eight stained preparations examined in Europe, one and a half years later, two trypanosoma found after a long search.

On June 27th the horse is already better; on the 28th it is quite well again, and, according to news received by letter later on, it remained in good health.

CASE 4.—Horse, the property of Mr. Schlapezek, 4 years old; has been in Little Popo two and a half or three years; formerly always healthy. His owner had made a four weeks' tour, concluded two days ago, to Topli in the hinterland of Togo. The tour was

only interrupted by a three days' stay on the coast. During the tour the horse and his master had frequently been wet through. For eight or nine days the horse had appeared unwell, was feverish, and off its feed, and did not drink much. No alteration of the urine and stool was remarked. For a few days a slight swelling of the hind legs had been observed, and there was a ribbon-like thickening on the abdomen.

On examination the horse was found to be in poor condition. Temperature 39.6°. The hind legs were found to be oedematously swollen. Blood examination of the living blood negative. The examination was very brief as the home-going steamer awaited me. In permanent preparations examined later, a very few trypanosoma were observed. Later fate of the horse is unknown.

From this observation it may be gathered that animals may become infected with tse-tse not only in the hinterland of Togo, but also in the immediate neighbourhood of that strip of land in Togo that lies between the lake and the sea near by. Now as the coastal strip of Upper Guinea possesses similar climatic and geological conditions, as also the same fauna and flora, it may be reasonably conjectured that the coastal region of the whole of Upper Guinea, with its hinterland, is infected. In regard to the details as to clinical symptoms, epidemiology, &c., the reader is referred to the literature.‡

It is well known that, after a stage of incubation of nine to twelve days, the disease breaks out either acutely, so that the animals perish under the appearance of rapidly increasing weakness, emaciation and anæmia, or a chronic stage sets in, in which the parasites periodically disappear from the blood, reappearing from time to time. The infected animal may then die after an illness of several months. Spontaneous cure may, according to R. Koch, set in in East Africa, but is of rare occurrence. The histories of disease here set forth show that both forms occur in Togo, as also spontaneous cure (vide Case 4). As to the cure of the inoculated kid, it cannot be brought forward as a test case, as the infection was not natural. It may, however, be noted that a horse and dog inoculated by Schilling both died from this artificial infection. According to Schilling, the oedema of the scrotum, the glans penis, the extremities, and the swelling on the abdomen, may be entirely lacking in chronic cases. Case 1 of the terrier bitch shows that in the most acute cases this symptom may be absent, at least in dogs. As Schilling found parasites in the blood of the inoculated animals six days after subcutaneous inoculation, and I only confirmed parasites in the kid eight or nine days subsequently, it is possible that in my experiments the far smaller quantity of blood injected, $\frac{1}{4}$ ccm., had something to do with this. Whereas Schilling did not find splenic tumour, this

† Dr. Schilling in his essay mentions two kinds of ticks in Togo: a large grey tick, such as I also had found, and a smaller, flat kind, with dark brown and red drawings on the back. It is probable that the latter are a younger stage of the former. The larvæ, nymphæ, males, and young females are essentially different from the large females seen in the former kind.

§ Hans Ziemann on "Blood Parasites in Temperate and Tropical Malaria," *Centralbl. f. Bakt. u. Paras.*, 1896, Nos. 18, 19. Also "Report on Malaria," &c., *Deutsch. med. Woch.*, 1900 (Reply to Dionisi).

|| Bruce, "Further Report on the Tse-tse Fly Disease or Nagana in Zululand." London. 1897. Robert Koch, "Travel-reports on Rinderpest, &c., Tse-tse or Surra Disease, &c." Berlin, 1898. Plimmer and Bradford, *Centralbl. f. Bakt. u. Paras.*, 1899, Part I. p. 440. Lingard, "Report on Horse Surra, Bombay," cited from Dolfein, "Protozoa." (Lingard's work is not yet published.)

symptom was undoubtedly present in Case 1. This possibly has something to do with the material used for observation. In regard to this and other small differences in observation, as, for instance, the ecchymoses, comparisons must be made with original matter (under the description of the parasites, where further differences will be noticed). The swelling of the testicles, which Schilling mentions as a sign of disease, I have not seen. I may, however, mention that in Cameroon, in 1894, while making enquiries on the diseases of animals on the West Coast of Africa, I heard of "malaria" in horses in Togo which commenced with scrotal swelling. Now this "malaria" was nothing but tse-tse disease (see H. Ziemann, *Ueber Malaria u. andere Blutparasiten*, 1898, p. 98). Schilling likewise observed the peculiar staggering in a sick pony, only this animal did not fall down as did the terrier mentioned, which fell down again and again as soon as it was stood up.

NAME AND ETIOLOGY.

Long before the discovery of tse-tse disease, peculiar parasites, the *Trypanosoma lewisi* (Kent), were found in rats, and they have recently been excellently described by Rabinowitsch and Kempner, Senn and v. Wasielewski. The parasites are slender, exceedingly lively flagellates, with a long anterior cilia of the same length as the cellular body itself, and possessing an undulant membrane. In preparations stained by the method to be described below, a thick, round grain of chromatin is observed in the anterior part, and a smaller one in the posterior part, conjectured to be the root of the cilia, from which the undulant membrane originates, and which in front forms the anterior process.

Propagation, according to these authors, occurs by means of longitudinal or transverse division, and by segmentation through division into numerous rosette-shaped shoots. They are mostly harmless parasites of the rats which are transmitted by fleas. These are morphologically different from the actual tse-tse parasites, as has been proved by Koch's inoculation experiments. Tse-tse disease or nagana has long been known in South and East Africa, as also a very similar disease, especially affecting horses, camels, elephants, and buffaloes in the East Indies. It is called *surra*, and, according to Evans, is transmitted by large flies; and is named *dourine* in Algiers, the south of France, and the north of Spain, where it occurs in horses and asses. These diseases are not transmitted by the tse-tse fly, but by other stinging insects. Nocard considers the three diseases to be one and the same, and Schilling is inclined to the same opinion, and therefore calls the disease in animals in Togo "surra." Doubtless the three diseases and their exciter are intimately related. In the meantime the exciters of the hæmoglobinuria of cattle in Germany are also morphologically very similar to those of Texas fever, as the author was able to prove,* and

yet, for epidemiological reasons, it is best to divide the two diseases, as the hosts of the germ of disease are two different kinds of ticks, which transmit the *Piroplasma bigeminum*.

The exciter of the East Indian "surra," the *Trypanosoma Evansi*, is asserted to be somewhat longer than the exciter of tse-tse. The trypanosoma of "dourine" likewise exhibits diversities, as it is not transferable to ruminants, as proved by experiments on animals. Schilling, moreover, proved that the tse-tse fly, which is found in Togo also, having fed on sick animals, transmits the disease, I therefore suggest that the name "tse-tse" be used.

OBSERVATION OF THE TOGO TSE-TSE PARASITES IN THE LIVING BLOOD.

As in all blood examinations in man and beast it is usual to take the blood to be examined from a wound on the ear, the examination in the suspended drop is only to be recommended for the purpose of demonstrating the duration of life of the parasite, or the changes undergone on adding various materials. It is more advisable for this purpose to keep the blood in capillary tubes; I thus kept the trypanosoma of the kid's blood (in the refrigerator of the steamer) alive for days.

Usually the specimens are made in the same manner as preparations of malarial blood.

One may then observe, corresponding to the case, a larger or smaller number of slender, excessively motile trypanosoma, having a flagellum at the anterior sharply-pointed end, and which is about one-third to one-fourth the length of the protoplasm. The length of the parasite varies considerably in the very motile forms, averaging 16-18-20 μ , the breadth about 2 μ . There were, however, also smaller parasites $11\frac{1}{2}$ to 14 μ in length. The movements, as Schilling also observed, were more violent at the flagellated end, so much so that the blood corpuscles are whipped hither and thither by the motions. The movement as a rule is serpentine; sometimes the two extremities bend towards each other and then spring apart, and then the serpentine motion is taken up again. When a great many parasites are in the preparation, as was the case in the dead terrier, where in every field of vision (Leitz, ocular 1 and immersion $\frac{1}{12}$) there were at least forty to fifty trypanosoma wriggling about, it looked like a swarm of gnats, for parasites and blood corpuscles were all on the move. A study of details under such circumstances is impossible. A preparation containing fewer parasites is more suitable, especially when their movements are curtailed by the addition of certain liquids. The motility was least in the broader forms, the greatest breadth at the moment of the least motion being about 4 μ . In such forms, which were somewhat rare in my preparations, the flagellum was somewhat shorter and the posterior end was rounded off. The protoplasm itself reminded one of the appearance of *Trichomonas vaginalis*.

Sometimes parasites were observed in which both ends were pointed. Their motility, however, was but slight.

The bodies of the trypanosoma are not actually

* H. Ziemann, *Ueber Lomadiera eine Art ausserst verbreiteten Texas fieber in Venezuela*. *Deutsch. Med. Wochenschr.*, 1902, Nos. 20 and 21. (Translation in *JOURNAL OF TROPICAL MEDICINE*, August 1, 1902.) *Ueber das endemische Vorkommen der seuchhaften Hämoglobinurie der Rinder in Deutschland*. *Deutsch. Med. Wochenschr.*, 1901, No. 1.

hyaline but finely granulated. In fresh preparations several light refracting particles are sometimes observable in the body, a larger, mostly less sharply circumscribed spot in the anterior part, mostly on the border between the first and second quarter of the length. Quite near the posterior extremity there were besides one or two distinctly circumscribed light refractory spots measuring about $\frac{3}{4}$ μ .

The process of division so graphically described by Schilling was only observed in stained preparations.

(To be continued.)

Reviews.

THE PRACTITIONER'S GUIDE. By J. Walter Carr, M.D. Lond., F.R.C.P.; T. Pickering Pick, F.R.C.S.; Alban H. G. Doran, F.R.C.S.; Andrew Duncan, M.D., B.S.Lond., F.R.C.S., M.R.C.P. London, New York and Bombay: Longmans, Green and Co., 1902, pp. 1,107.

The text of this eminently practical book is arranged in dictionary form, and is therefore available for ready reference. The material has been brought within the scope of a single volume, but so carefully judged, balanced and sifted is the profusion of literature to which modern medical, surgical and gynæcological knowledge has attained, that ample justice is done to every branch of the subject essential to those engaged in the practice of medicine.

Tropical medicine is ably dealt with by Andrew Duncan, M.D., Joint Lecturer on Tropical Medicine at the London School of Tropical Medicine. Dr. Duncan handles the larger subjects appertaining to tropical diseases in a masterly fashion, and with a grasp which is to be highly commended. Considering the enormous number of old tropical residents and travellers who return to this country and who come under the care of medical men in quite remote parts of the country, a concise and succinct account of the more prevalent tropical ailments is a necessity. This Dr. Duncan has successfully accomplished. To the young practitioner also, starting for the Tropics, he will find in the pages of "The Practitioner's Guide" exactly what he wants, namely, a thoroughly up-to-date account of the diseases he will have to treat in the daily routine of practice. It will be observed that, unlike other works in medicine, "The Practitioner's Guide" includes the ordinary surgical operations and the diseases of women. A more useful hand-book for the practitioner has never been published.

THE SO-CALLED "SPOTTED FEVER" OF THE ROCKY MOUNTAINS. A Preliminary Report to the Montana State Board of Health. By Louis B. Wilson, M.D., Minneapolis, and Wm. M. Chowning, M.D., Minneapolis. *The Journal of the American Medical Association*, July 19th, 1902. "Spotted fever," "blue disease," or "black fever," are the various local names given to a peculiar disease

which has been known for about thirty years over limited portions of the States of Montana and Idaho in North America. The persistence of the disease in Montana, along the eastern foothills of the Bitter Root Mountains, led the Montana State Board of Health to undertake a special investigation as to its etiology and pathology. The investigation was entrusted to Drs. Louis B. Wilson and Wm. M. Chowning, of Minneapolis, who have carried it out most satisfactorily. Previous to their report, the only article on "spotted fever" in a medical journal was that by Dr. E. E. Maxey in the *Portland Medical Sentinel*, for October, 1899.

Dr. Maxey's paper describes cases in Idaho, mostly along the southern foothills of the Bloise Mountains.

In Montana, the disease is restricted to the western slope of the Bitter Root River valley. This valley is about 3,500 feet above sea-level, it has a mild climate, and is populated by fairly well-to-do ranchers from Missouri, Georgia, and the Carolinas.

Spotted fever is, as a rule, a very grave disease, with a high death-rate of 70 to 80 per cent. It attacks any age and either sex. It is definitely limited in its geographical distribution. In the State of Montana, while prevalent along the eastern slopes of the Bitter Root Mountains, it is sharply cut off from the eastern side of the valley by the Bitter Root River.

It is confined entirely to one season of the year, namely, spring. The earliest recorded case began on March 17th, and the latest about July 20th, but most cases occur between May 15th and June 15th.

According to Dr. Maxey, the disease appears to be more malignant in some localities than it is in others, and in one year more than in another. The disease is not contagious.

Symptoms.

The symptoms of spotted fever are characteristic of specific infection. Most cases are preceded by a short period of malaise, then follows a well-marked chill recurring at irregular intervals, though with decreasing severity, throughout the attack. At the onset, there is a severe aching in the bones and muscles with pains in the back and joints. The patient is usually very weak, but restless and headache may be severe. The skin is dry and the tongue is thickly coated. The coat at first white becomes brownish as the fever increases, while the tongue becomes dry and cracked. Indeed, the whole facies in these respects is very like typhoid.

After the initial chill, the temperature may reach 103° to 104° F. on the second day. It gradually increases and reaches its maximum in from five to seven days, when it may register from 105° to 107° F. Usually a slight evening increase and morning decrease is noted. Where recovery occurs it usually is by lysis, much as in typhoid. The diminution of the fever begins about the end of the second week and reaches normal about two weeks later.

A characteristic eruption appears on the skin from the second to the fifth day after the chill, it begins about the wrists and ankles, or on the back. It then extends over the entire body, covering the scalp, the palms of the hands and the soles of the feet. The abdomen usually is the last surface involved. As a rule, the eruption reaches its maximum in about two days, but sometimes it spreads very rapidly. At first

it consists of circular rose-coloured spots varying in size from one to five millimetres in diameter. These macules are not elevated; they disappear on pressure, but quickly reappear. They are sometimes tender to the touch; very soon they assume a dark blue or purplish colour, they increase in size and do not disappear on pressure. The confluence of the extending macules often gives a mottled or marbled appearance to the skin, especially in the dependent portions, but in some cases the eruption at no time becomes confluent, and only small brownish or purplish petechiæ may be present, giving a speckled appearance, which has been likened to that of a turkey's egg. The skin is not only covered with spots, but is also somewhat jaundiced; this may be quite marked in the conjunctivæ. About the third week desquamation begins. The spots fade as the fever subsides, but may not entirely disappear for weeks or months after convalescence is established. In some cases the skin may become gangrenous on the elbows, fingers, toes, lobes of the ears, &c. Constipation is usually present from the beginning. About the beginning of the second week nausea and vomiting develop and continue in fatal cases to the end. The liver is somewhat enlarged. The spleen is uniformly enlarged and tender on palpation. The urine is usually reduced in amount and highly coloured. A small amount of albumin, and both granular and blood casts were found in all cases examined. In all severe cases, more or less œdema of the face and extremities is present. The œdema may be marked and may appear as early as the third day of the disease. A low, muttering delirium as in typhoid fever is present in severe cases, the patient being but partly rational. The respiration rate is frequently out of all proportion to the temperature; sometimes it reaches 60° per minute in the adult, though ordinarily it does not run above 36° per minute. The pulse at the onset is usually full and strong, but gradually becomes more and more rapid, while it loses in volume and strength. In fatal cases in adults, it may reach 150° per minute some days before death. The red blood count in five cases examined was 4,100,000, 4,200,000, 4,300,000, 4,500,000, 4,600,000 respectively. There was a slight increase of leucocytes, from 12,000 to 13,000, in the four cases examined. Blood, when removed for examination, appears to be somewhat darker than normal, as well as somewhat less fluid. In five cases examined during various stages of the disease the hæmoglobin was 50 to 60 per cent. Freshly-drawn blood, when examined with the 1—12 oil immersion objective shows peculiar hæmocytozoa sparingly in the erythrocytes.

Morbid Anatomy.

Six autopsies were made. The following lesions were found: The skin was covered with a petechial rash, and in all cases presented small wounds due to tick bites. The lungs showed hypostatic congestion. The epicardium usually contained a few petechial hæmorrhages near the base of the left ventricle. The myocardium was softened. The right ventricle was filled with dark fluid blood; the left was almost empty or contained only a small clot. The spleen was enlarged, being from three to three and a half

times its normal weight. The capsule was distended and thinned. On section the tissue was found dark red and so soft as to be in most cases diffident. The stroma was not increased in amount, but it was engorged with blood cells and leucocytes. The omentum covering the spleen was usually congested. The liver was slightly enlarged, pale in colour and of normal consistence. Microscopically it showed pronounced fatty degeneration. In all cases one or both kidneys showed small sub-capsular hæmorrhages on the ventral surface. The capsule stripped readily. The cortex on section was congested. The meninges of the brain and spinal cord showed a slight congestion, apparently hypostatic.

Etiology.

Drs. Wilson and Chowning found in the blood of patients suffering from spotted fever an unpigmented hæmocytozoal parasite, probably belonging to the genus *Piroplasma*, and resembling very much the micro-organism of Texas fever, but it is larger, and in its larger forms exhibits active amœboid movements. In the peripheral circulation, probably not more than one erythrocyte in five hundred is usually infected, but in the congested capillaries in the tissues removed at autopsy from 1 to 5 per cent. of the erythrocytes contain parasites. The majority of infected cells are in the lung, spleen, liver and kidney. In these organs many infected erythrocytes are included in phagocytes.

The organism of spotted fever varies greatly in size, form and staining reaction at various stages of its development. The smallest forms (phase 1) when seen in fresh (unstained) preparations, are intracellular, ovoidal bodies, about one micron in thickness, and one and a half to two microns in length. A few, but certainly not nearly all, of these bodies are stained by Loeffler's methylene blue. When so stained the smaller extremity takes up more of the stain than does the larger extremity, which remains but faintly blue even after prolonged staining. Perhaps one in ten of the red cells which are infected with this form of organism contains two instead of one of the bodies. When the organisms are in pairs within a cell, their long axes usually lie in the same straight line, though they may be placed at an angle as acute as sixty degrees. Their lesser extremities are often toward each other, though distinctly separated by a small interval. These bodies show no evidence of amœboid movement, but they have occasionally been seen to change their position within the erythrocyte.

Several gradations of size, in both single and paired organisms, have occasionally been observed up to phase 2. This phase is marked by an organism solitary within the red cell, and usually ovoidal in form, though it may be elongated, ellipsoidal or spheroidal. It is from two to three microns thick and three to five microns long. This type in freshly-drawn blood frequently exhibits active amœboid movements. The movements consist in the elongation of the organism, the projection and retraction of pseudopodia, and the assumption of the ovoidal form. The ovoidal form, however, appears to be lost shortly after death, since although many of this form were present in the tissues from an autopsy made three hours after death, but very few were present in the tissues from other autopsies made seven and eight hours after death.

In these tissues the organisms were almost all spheroidal. In the tissues removed at autopsy the parasites are metachromatic to methylene blue.

In the freshly-drawn blood are sometimes found bodies measuring about 0.5 to 1 micron in diameter and exhibiting active Brownian movement. Possibly they represent phase 3. They are found occasionally in pairs, and these resembling diplococci. They vary much more in size than do cocci of one species, and sometimes, also, are ovoidal in shape. They stain but faintly with Loeffler's methylene blue, and indeed in all respects resemble the smallest intracellular forms except that they are smaller, extracellular, less numerous and more diplococcus-like. In direct cover-slip preparations made from the blood at autopsy no extracellular forms have as yet been found.

Drs. Wilson and Chowning made some inoculation experiments and obtained positive results in two rabbits. These rabbits, on the day following inoculation, showed a rise of temperature of 1° C. At this time intracellular organisms were found in the blood of both rabbits, and continued present in specimens collected on successive days for two weeks.

Drs. Wilson and Chowning believe that the parasite of spotted fever is probably conveyed to man by ticks. They base this opinion not only on the analogy of the closely allied *Piroplasma bigeminum* which is transmitted by the cattle tick, *Rhipicephalus annulatus*, but on the very suggestive fact that all the patients (eleven) which came under observation during their investigations had been bitten by ticks. In three cases a history was given of a single severe tick bite two, three and five days respectively, before the onset of the disease. Besides, there is no evidence whatsoever to prove that spotted fever can be transferred in a direct manner from man to man; likewise, there is no evidence to prove that it may be transmitted by the means of water or food. The occurrence of the disease in isolated cases in a region sharply limited on one side by a river, would indicate the conveyance of the germ to man (if by any animal whatsoever) by a temporarily parasitic animal which travels slowly and not widely, and which is not carried far by the wind, &c. The tick answers this description. Moreover, the seasonal limitation of spotted fever coincides most admirably with the period of the year in which ticks are active.

The extreme isolation of cases of spotted fever, their occasional development in localities removed many miles from the site of any previous case, and the long period existing between the death or convalescence of the last case of any one year before the development of the first case in the following year, would point to the possibility of the red blood cells of some of the lower warm-blooded animals being the normal habitat of the parasitic protozoon in that stage of its life-cycle not passed within the body of some arthropod. Of the animals within the infected region the common gray gopher would, according to Drs. Wilson and Chowning, best fulfil the conditions of such a parasitism.

Drs. Wilson and Chowning are now attempting to obtain data which shall confirm or demolish the above hypothesis.

DIE MALARIA DER AFRIKANISCHEN NEGERBEVÖLKERUNG BESONDERS MIT BEZUG AUF DIE IMMUNITÄTSFRAGE (The Malaria of the African Negro Population, more especially in Relation to the Question of Immunity). By Dr. Albert Plehn. Jena: Gustav Fischer, 1902.

This interesting work exhibits the scientific accuracy and close study with which we are familiar in the writings of this author, and his statements are supported by practical observations. Dr. Plehn states that in both the children and adults of the negro population malarial parasites are present in the blood, independently of their state of health. In nearly all negro infants, even of the tenderest age, numerous malarial parasites are found in the blood without detriment to the health and without causing fever; on the other hand, however, attacks of fever sometimes supervene without demonstration of malarial parasites in the peripheral blood.

According to Plehn's opinion the strict division of the various parasites into three or four definite forms is untenable, as according to the particular condition under which it develops the parasite is capable of assuming various forms, or, indeed, may pass from one form to another, determined by the divergent development of the vacuole, nucleus and plasma.

These observations are demonstrated by a series of excellent coloured plates illustrating numerous observations on the forms of the parasites of quartan, tertian and irregular fevers.

Plehn considers Koch's theory on the extermination of malaria by destroying the malarial parasites in man as untenable, as a quinine prophylaxis is founded on the parasitic condition of the blood in inhabitants of the tropics, and fever sometimes exists without the demonstration of parasites in the peripheral blood and *vice versa*; there is also the possibility of infection of the mosquitoes to be taken into account.

News and Notes.

MR. HENRY S. WELLCOME has issued invitations for a dinner to be given to Dr. Andrew Balfour, of Edinburgh, previous to his departure for Khartoum, where he is to take up his duties as Director of the Clinical and Bacteriological Research Laboratories in the Gordon's Residential College. Dr. Balfour leaves for Egypt on December 11th.

THE Copley medal of the Royal Society has been awarded to Lord Lister "in recognition of the value of his physiological and pathological researches in regard to their influence on the modern practice of surgery."

It is announced that Professor R. Koch intends visiting South Africa for the purpose of investigating the widespread disease amongst cattle prevalent there.

THE SLEEPING SICKNESS COMMISSION IN UGANDA.

Dr. Low, Craggs Research Scholar, London School of Tropical Medicine, is on his way home from Uganda. It will be remembered that Dr. Low, in conjunction with Dr. Castellani and Dr. Christy, was sent out some months ago by the Royal Society and Foreign Office to investigate sleeping sickness in Uganda. Although Dr. Low has finished his work in connection with the pathology of the disease, Dr. Castellani and Dr. Christy are remaining behind for a time, the former to continue his bacteriological work, whilst Dr. Christy is to visit other districts, and intends returning to England after traversing the region of the Upper Nile. No official report is as yet to hand, but a statement has been circulated in the press that the members of the Commission hold that the disease is bacterial in its nature and not of filarial origin, as was at one time suspected.

A LEPROSY ENQUIRY.

Mr. Jonathan Hutchinson, F.R.S., starts for India on December 22nd, in the hope of being able to remove, by enquiries on the spot, certain objections to his hypothesis that the consumption of badly cured fish is the cause of leprosy. His itinerary is to be Ceylon, Madras, Calcutta, Assam, and finally Northern India.

Whether Mr. Hutchinson's theory of the origin of leprosy is believed or not, we are sure that so astute an observer will add considerably to our knowledge of the distribution of the disease. His theory regarding the etiology of leprosy and his opinion in regard to the futility of complete segregation as a means of eradicating leprosy are so well known, that we expect Mr. Hutchinson will devote his attention almost solely to these two arguments. Whilst regretting this in the main, we can only see that good can come of the enquiry, for as yet, Mr. Hutchinson's is the only working hypothesis advanced concerning the etiology of leprosy, apart from its bacterial nature, that we have before us. We wish Mr. Hutchinson a safe and scientifically successful voyage.

COLONIAL TRAINING FOR WOMEN.

The Horticultural College at Swanley, Kent, is about to commence a course for the training of women for colonial life. The course will occupy about a year, and special attention will be paid to horticulture, dairy work, and out-door occupations. In addition to these subjects the syllabus of instruction includes seed sowing, fruit packing, jam making, fruit bottling, cow keeping, carpentering, household management, colonial hygiene, and native languages.

SIR WILLIAM MACGREGOR, K.C.M.G., GOVERNOR OF LAGOS, IN LIVERPOOL.

At Liverpool, on November 17th, the African Trade Section of the Liverpool Chamber of Commerce entertained H.E. Sir William Macgregor, at dinner. Sir A. Jones, K.C.M.G., occupied the chair, and whilst proposing the health of "the Governor of Lagos," stated that a great improvement had been made in the sanitary condition of West Africa, that Liverpool had contributed largely towards that desirable end, and that Liverpool had started the Tropical School and set the world on fire. A deep debt of gratitude was due to Sir

William Macgregor for what he was doing in West Africa. In the course of his reply, Sir William Macgregor said that in trying to improve sanitation in Lagos efforts should not be confined to benefit persons of one race or colour, but that natives and Europeans should be considered together, and he had steadily and consistently been opposed to the idea of separating Europeans from the natives, in order that the former should not contract malaria from the natives. He firmly believed that the extermination of malaria could only be looked for by carrying Europeans and natives forward in one line. Lagos had become the outlet of the vast area of Northern Nigeria, and could malaria be reduced a great future was in store for the colony. Mr. John Holt, in his speech, said that Major Ross would live in Liverpool annals, and in the annals of the nation, as one of the benefactors of the human race.

FUNDS FOR THE LIVERPOOL SCHOOL OF TROPICAL MEDICINE.

The Lord Mayor of Liverpool proposed the toast of the Liverpool School of Tropical Medicine at the dinner to Sir William Macgregor, on November 17th. Mr. W. Adamson, Vice-Chairman of the School, in replying, said that Professor Boyce, by his exertions, enabled him to announce the fact that a chair in the University College had not only been founded but endowed by special contributions amounting to over £10,000; of this total Sir Alfred Jones had contributed £5,000, and the Hon. R. B. Blaize, of Lagos, £500. He hoped the chair would be called the Sir Alfred Jones Chair of Tropical Medicine and Parasitology, and that Major Ross would be elected as the first professor.

Sir Alfred Jones stated that he had received a telegram from Mr. Chamberlain congratulating Liverpool on the great success of the School, and also announced he had received another message from the King of the Belgians, in which His Majesty states he was willing to contribute £500 towards the expenses of sending representatives of the Liverpool School of Tropical Medicine to the Congo.

Current Literature.

A NEW THEORY OF THE WAY QUININE ACTS.—Dr. A. F. A. King, U.S.A., suggests that the curative effect of quinine may be due to fluorescence producing violet rays of light in the blood. His opinion is based on the supposition that the malarial parasite will not sporulate in the dark; that the light it has in the blood is red; and since it has been demonstrated that *amœba proteus* streams in the presence of red light and ceases to stream in violet light, it has been assumed that the same may be true of the malarial *amœba*.—*Amer. Journ. Med. Sc.*, June, 1902.

UNDUE EFFECTS OF QUININE ADMINISTRATION.—M. A. Martinet draws attention not only to the aural disorders attributed to quinine, but states that in old people the drug is apt to cause irritability of the

bladder. He is not of opinion that quinine causes uterine contractions; it may, however, induce vomiting, cutaneous eruptions, and in poisonous doses cause convulsions, loss of consciousness, collapse, cyanosis, and suppression of the urine. Dr. Martinet, however, states that these deleterious effects may be combated by aperients, diuretics and stimulants.—*Presse Médical*, April 19th, 1902.

CONTRIBUTION TO THE ANOPHELES FAUNA OF WEST AFRICA.—Dr. Hans Ziemann had the opportunity of collecting and examining numerous mosquitoes in Duala, Victoria, near the Cameroon mountains, in Togo and suburbs, and in Monrovia. The specimens were forwarded to Germany for identification by Dr. Enderlein.

The author observed that the *Anopheles* everywhere were infinitely more numerous during the rainy season than in the dry season.

A new *Culex* (not yet classified) was found on the Cameroons at an altitude of 1,600 metres, but no *Anopheles* were found in Buča (the health resort of Cameroon), at an altitude of 900 metres. They were numerous in the Molive plantation, 220 metres above the level of the sea, but were scarce in the Boana plantation, 260 metres above sea-level.

It is a curious fact that the *Anopheles* in Victoria suck blood only a few hours after they have emerged from the pupa; but in Togo they could only be made to bite twenty-four or even forty-eight hours after.

The following varieties are found:—

Anopheles costalis, Loew. Frequent in Cameroon, particularly during the rainy season, on the upper reaches of the Wuri River, in Victoria, in Togo, Monrovia, and Upper Guinea.

Anopheles funestus, Giles. In Victoria, frequent in the huts of the natives during the rainy season.

Anopheles Pharoensis, Theobald. Frequent on the upper reaches of the Wuri River near Duala, in September in Victoria, and in Lome and Togo in Upper Guinea.

Anopheles Ziemanni. A new species, described by Grünberg (*Zoolog. Anzeiger*, July 21st, 1902), in large numbers on the Wuri River, but were never observed in Duala or in the neighbourhood.

A. costalis, *funestus*, *Ziemanni*. All these varieties transmit malaria.—*Archiv für Schiff's- und Tropen Hygiene*, October, 1902.

TREATMENT OF TARANTULA BITE.—Dr. J. S. Boyers reports the case of a healthy man, aged 52, who was bitten on the forefinger by a gravid female tarantula five inches in length. Dr. Boyers arrested the circulation of blood in the finger with a narrow bandage. He then made a crucial incision to the bone over the site of infection and held the finger under the tap, exposing it thoroughly in the running water for about one minute, and applied a saturated solution of potassium permanganate, working it thoroughly into the wound. The symptoms were as follows: Complexion of an ashen hue. The extremities cold and bathed in perspiration. The pupils slightly dilated. Hearing considerably impaired. The constitutional treatment consisted first of $\frac{1}{30}$ grain of strychnine with $\frac{1}{100}$ grain of nitroglycerin hypodermically, and during the first hour and a half after the bite he was given in

all, by mouth and hypodermically, in small doses frequently repeated, $\frac{1}{15}$ grain of strychnine, $\frac{1}{30}$ grain of nitroglycerin, $\frac{1}{8}$ grain of atropine, $\frac{1}{4}$ grain of morphine, 1 oz. of aromatic spirits of ammonia, and 6 oz. of good brandy, besides using artificial heat. The bowels, kidneys, and skin were kept active. The patient recovered in four or five days.—*Fort. Wayne Med. Journ.*, September, 1902.

Pernicious Anæmia.

Dr. J. N. Danforth obtained good results from a combination of—

| | | | | | |
|---|-----------------------|----|----|----|---------|
| R | Liq. Potas. Arsenitis | .. | .. | .. | 5 iiss. |
| | Acid Phosphate | .. | .. | .. | 3 iij. |
| | Ext. Bone-marrow | .. | .. | .. | 3 viij. |

M. S. Two drachms after each meal.

—*Medical Bulletin, U.S.*

| | | | | | |
|---|---------------------|----|----|----|---------|
| R | Fowler's Solution | .. | .. | .. | 3 iss. |
| | Acid Phosphate | .. | .. | .. | 3 iij. |
| | Bone-marrow Extract | .. | .. | .. | 3 viij. |

M. S. Dessertspoonful after each meal.

—*Medical Times and Hospital Gazette*, June 28th, 1902.

Alcoholic Cirrhosis of the Liver.

One of the best diuretics is—

| | | | | | |
|---|-----------------|----|----|----|--------|
| R | Juniperi Fruct. | .. | .. | .. | 10 gm. |
| | Infuse in | | | | |
| | Aq. Bul. | .. | .. | .. | 200 „ |

Add

| | | | | | |
|--|-----------------------|----|----|----|------|
| | Potass. Nitrat. | .. | .. | .. | 2 „ |
| | Potass. Acetat. | .. | .. | .. | 50 „ |
| | Scillæ Oxymel | .. | .. | .. | 30 „ |
| | Syr. des Cinq Racines | .. | .. | .. | 30 „ |

M. S. To be taken in four or five doses during the day.

A Useful Hepatic Stimulant.

| | | | | | |
|---|-----------------|----|----|----|---------|
| R | Pulv. Rhei | .. | .. | .. | gr. ij. |
| | Pulv. Ipecac. | .. | .. | .. | gr. ½ |
| | Sodii Bicarb. | .. | .. | .. | gr. v. |
| | Tinc. Nuc. Vom. | .. | .. | .. | ℥ v. |
| | Ol. Menth. Pip. | .. | .. | .. | ℥ 1/20. |

M. ft. tab. No. i.

Cramps, Colic and Diarrhœa.

| | | | | | |
|---|---------------------|----|----|----|------------|
| R | Morphin. Muriat. | .. | .. | .. | gr. 1/12. |
| | Ex. Cannabis Indic. | .. | .. | .. | gr. 1/8. |
| | Nitroglycerin | .. | .. | .. | gr. 1/600. |
| | Ext. Hyoscyami | .. | .. | .. | gr. 1/4. |
| | Oleoresin. Capsici | .. | .. | .. | gr. 1/20. |
| | Ol. Menth. Pip. | .. | .. | .. | ℥ 1/20. |

For one tablet or capsule.

Dysentery.

| | | | | | |
|---|-------------------------|----|----|----|----------|
| R | Acidi Sulphurici diluti | .. | .. | .. | 3 ss. |
| | Tinct. Opii deodorati | .. | .. | .. | aa 3 j. |
| | Spir. Camphoræ | .. | .. | .. | aa 3 j. |
| | Tinct. Capsici | .. | .. | .. | aa 3 ss. |
| | Spir. Chloroformi | .. | .. | .. | aa 3 ss. |
| | Spir. Vini Gallici | .. | .. | .. | aa 3 ss. |

M. S. 3 j. q. 2—3 h.

Or—

| | | | | | |
|---|-----------------------|----|----|----|-------------|
| R | Magnesii Sulphatis.. | .. | .. | .. | 5 j. |
| | Acidi Sulphur. diluti | .. | .. | .. | aa 3 j. |
| | Tinct. Opii deodorati | .. | .. | .. | aa 3 j. |
| | Aq. Chloroformi | .. | .. | .. | q. s. 3 ij. |

S. This dose q. 3 h.

Typhoid Diarrhœa.

| | | | | | |
|---|-------------------|----|----|----|--------------|
| R | Acidi Carbolici | .. | .. | .. | gr. j. |
| | Ext. Opii | .. | .. | .. | gr. j. |
| | Bismuth Subnitrat | .. | .. | .. | gr. xviiiij. |

M. et ft. pil. No. vj. S. One t. i. d.

—*Medical Times and Hospital Gazette*, July 5th, 1902.

MALARIA.

EPILEPSY IN PERSONS SUFFERING FROM MALARIA.—The relation of malaria to epileptic seizures has recently been dealt with by Dr. Corsini. The author distinguishes between the attacks of epilepsy occurring during an attack of actual malaria fever and those which develops in persons who are merely the subjects of malaria. Corsini relates the case of a patient in whom, during the period of chill, epileptic convulsions almost invariably supervened. The paroxysm ran its natural course of chill, high temperature, and perspiration. The relationship of the epileptic seizure to the onset of the chill is difficult to gauge; that it is due to cerebral irritation caused by an accumulation of malarial parasites in the blood-vessels of the brain would appear untenable, seeing that it is only in æstivo-autumnal fever that such a condition exists. The mere mention, however, of the connection of epilepsy with malarial fever is an interesting feature, and may aid us in elucidating the etiology of epilepsy.—*Gazzetta degli ospedali e delle cliniche*, August 3rd, 1902.

THE MOST RATIONAL PERIOD TO ADMINISTER QUININE IN MALARIAL FEVER.—Dr. I. A. McSwain says that as the spores of the malarial parasite escape into the blood at the end of the stage of chill and rigor, quinine ought to be administered at that time, as thereby the newly-formed spores are exposed to the influence of the drug before they can re-enter the fresh red corpuscles of the blood.—*Southern Practitioner*, August.

PLAGUE.

THE PLAGUE, ITS "DIFFUSIVE TENDENCY"; HAFFKINE'S ANTI-PLAGUE VIRUS v. YERSIN'S SERUM.—Dr. T. D. Burch draws attention to Calmette's statement that persons treated, during the incubation period of plague, by Haffkine's serum would suffer from the disease when it developed in an aggravated form. Major Bannerman, I.M.S., in his reports states, however, that Haffkine maintains that inoculation with his vaccine is harmless in the incubation period of the disease, and can be safely employed for persons who have been in contact with those suffering from plague. Calmette advocates the use of Yersin's serum both before and after exposure to plague. Experience in India, however, refutes Calmette's contention, and the opinion at the present moment is greatly in favour of Haffkine's serum in preference to any other.—*New York Med. Journ.*, September 20th, 1902.

THE PRODUCTION OF IMMUNITY IN PLAGUE BY ANTI-PLAGUE VACCINE ADMINISTERED BY THE MOUTH.—Mercatelli Vincenzo, by way of dealing with the introduction of an anti-plague vaccine into the system by some other method than by subcutaneous injection, has been experimenting on guinea-pigs by administering a bolus of anti-plague vaccine by the mouth. He found that large doses caused gastro-enteritis, which sometimes proved fatal. Vincenzo found that one dose contributed a slight degree of immunity to plague in the animal experimented upon, and he is of opinion that with repeated progressive doses a further immunity might be obtained. Unfortunately, the author does not mention the dose of the vaccine; but in view of the refusal of many persons, native and European

alike, to vaccination or subcutaneous injection, it is to be hoped Vincenzo will continue his experiments.—*La Riforma Medica*, August 5th, 1902.

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Archives Russes de Pathologie, de Médec. Clinique et de Bacteriologie.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Brooklyn Medical Journal.
Caducée.
Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Esercito.
Hong Kong Telegraph.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
Journal of the American Medical Association.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Medical Record.
Medical Review.
Merck's Archives.
New York Medical Journal.
New York Post-Graduate.
Pacific Medical Journal.
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Revista de Medicina Tropical.
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Sei-i-Kwai Medical Journal.
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The Journal of Tropical Medicine.

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Original Communications.

A REPORT OF FOUR CASES OF MALARIA AT THE BRANCH SEAMEN'S HOSPITAL, ROYAL ALBERT DOCK, TREATED BY ARRHENAL.

Under the care of Dr. PATRICK MANSON, C.M.G., F.R.S., with a Commentary by C. W. DANIELS, M.B., M.R.C.S. Reported by W. G. ROSS, M.D., M.R.C.S.

THE immediate effects of quinine are uniformly markedly beneficial in cases of malaria. The action as regards the attack is rapid and certain, but unfortunately, unless the treatment is continued for a protracted period, recurrences are frequent. Any drug, therefore, that has as good an immediate effect and also prevents relapses and recurrences, would be most valuable. Dr. Armand Gautier, of Paris (*Archives de parasitologie*), claimed these properties for a new cacodylate called arrhenal and gave illustrative cases treated by this drug. Four cases of malaria have been treated at the Branch Seamen's Hospital by this method. A brief history of each case and temperature chart is appended.

CASE I.—W. R., aged 33, clerk. Last from Hong Kong. Admitted June 14th, 1902. Had malaria in Hong Kong for the first time on March 2nd, 1901; a severe attack; was in hospital three weeks; had several relapses and was invalided home on November 10th, 1901. Did not have any recurrence after that date until an attack on May 27th, 1902, which lasted until his admission on June 14th.

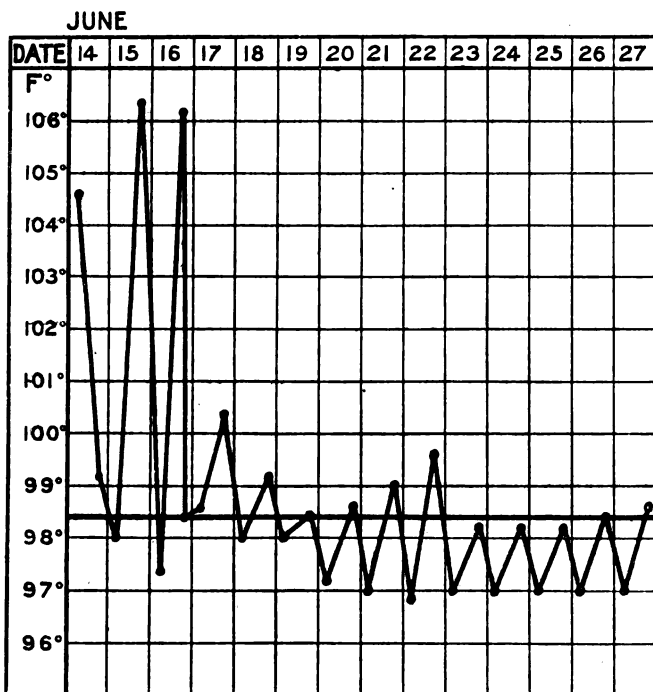
An examination of his blood showed a small quartan and a moderate double tertian infection. He had a daily rigor and temperature of over 106°. When the character of his infection had been ascertained, 9 grains of the bi-hydrochlorate of quinine were injected into the gluteus maximus daily for three days; his temperature fell to normal, and he was put upon 5 grains of the sulphate of quinine three times daily.

This he continued to take from June 19th until September 7th, when he stopped taking it. On the 9th he caught a cold travelling in a night train. He applied again for admission on September 15th, when his temperature was 103·8°. On the 16th there was no fever; on the 17th he had a rigor and a temperature of 106·2°. An examination of his blood showed a large infection of benign tertian parasites. This seemed a very suitable case for the use of arrhenal. On September 18th, 19th, and 20th, 5 centigrammes were injected intramuscularly, and on the 21st, 10 centigrammes. On the 22nd there was no fever, but the number of the parasites was such that fever was sure to occur. The patient was decidedly ill, and at 9.30 p.m. on that day an intramuscular injection of quinine was given. On the 23rd there was no rigor, but the temperature rose to 105·4°. On the 24th the temperature had become normal and the parasites were very few in number. An injection of quinine was given on the 22nd, 23rd, and 24th, and quinine was continued by the mouth during the rest of the patient's stay in the hospital. There were no parasites found in his blood on the 29th, and the patient was discharged on October 6th, there being no rise in temperature and no parasites in the blood; he was instructed to take quinine for three months.

Remarks.—This was a case in which the febrile attacks were known to yield readily to quinine, but in which even the prolonged use of quinine did not prevent recurrence of the disease. As the temperature chart and blood examinations show, arrhenal had no beneficial effect at all, whilst quinine had a rapid action.

CASE II.—C. B., aged 36, seaman. Last voyage from Delagoa Bay. Admitted September 22nd, 1902. Acquired malaria at Delagoa Bay in May, 1902. Had repeated relapses and was irregularly treated by quinine and phenacetin. Blood examination on admission showed benign tertian parasites, a moderate infection; spleen and liver very much enlarged. On the 23rd had a severe rigor and a temperature of

105.2°. On the 25th the temperature rose to 102.4° without rigor, and he was found to have two infections of benign tertian parasites. From the 26th to the 28th there was little or no fever and the parasites



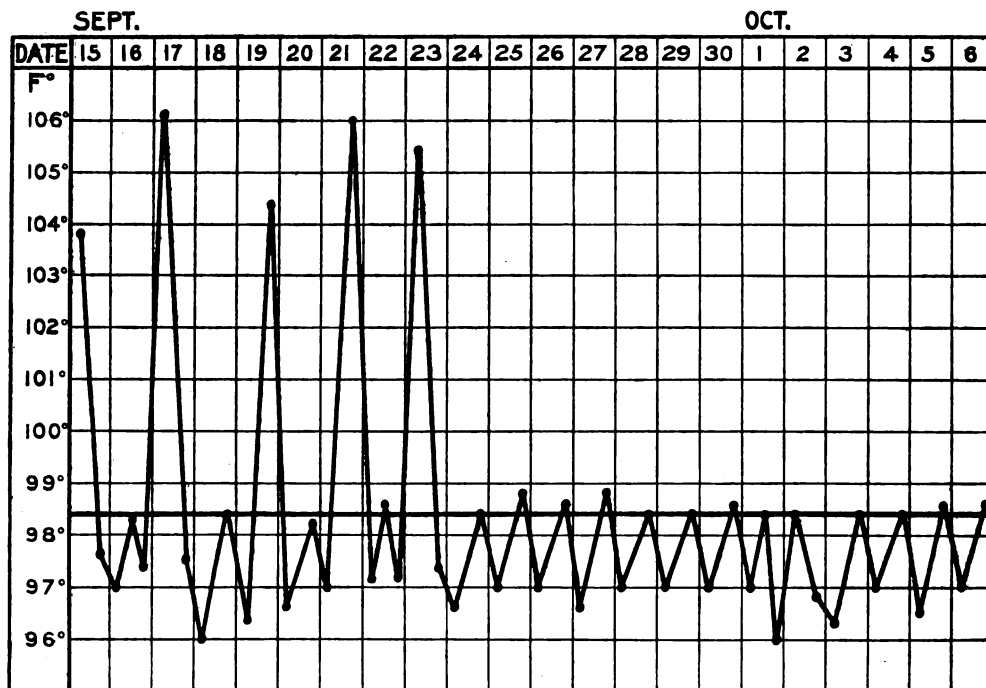
CASE I.

and a temperature of 104.8°. On October 1st, 5 centigrammes of the arrhénal were injected intramuscularly, and continued to be given in this way daily until the 3rd. On the 4th, 10 centimetres were given. The chart temperature showed an irregular rise, usually twice a day, to 105.2° and 105.6°. On the evening of the 4th the patient vomited and passed blood *per rectum*. It was decided to inject quinine at 2 a.m. on the morning of the 5th. At 11 a.m. on the same day the injection was repeated, when the temperature became normal. Five grains of quinine were given by the mouth three times a day afterwards. There were no parasites in his blood on the 6th.

Remarks.—Here we have a case in which warmth and rest in bed had a decided effect for a time, but arrhénal failed whilst quinine was effective.

The cases quoted by Dr. Gautier were of the so-called malignant tertian form of malaria. The failures in benign tertian were not conclusive. Two cases of malignant tertian were therefore treated as recommended by Dr. Gautier.

CASE III.—J. C., aged 33, seaman. Last voyage from Cuba. Admitted September 27th, 1902. Acquired malaria in Cuba in July, 1902. Had repeated attacks on board ship and was given quinine irregularly by the captain. Arrived home September 21st, and has had fever daily ever since. His temperature on the 27th was 104.4°, and a blood examination showed young forms of the malignant parasite. Crescents in addition to ring forms were found on October 1st. He had a daily temperature above 103° until October 2nd, when the arrhénal injections were



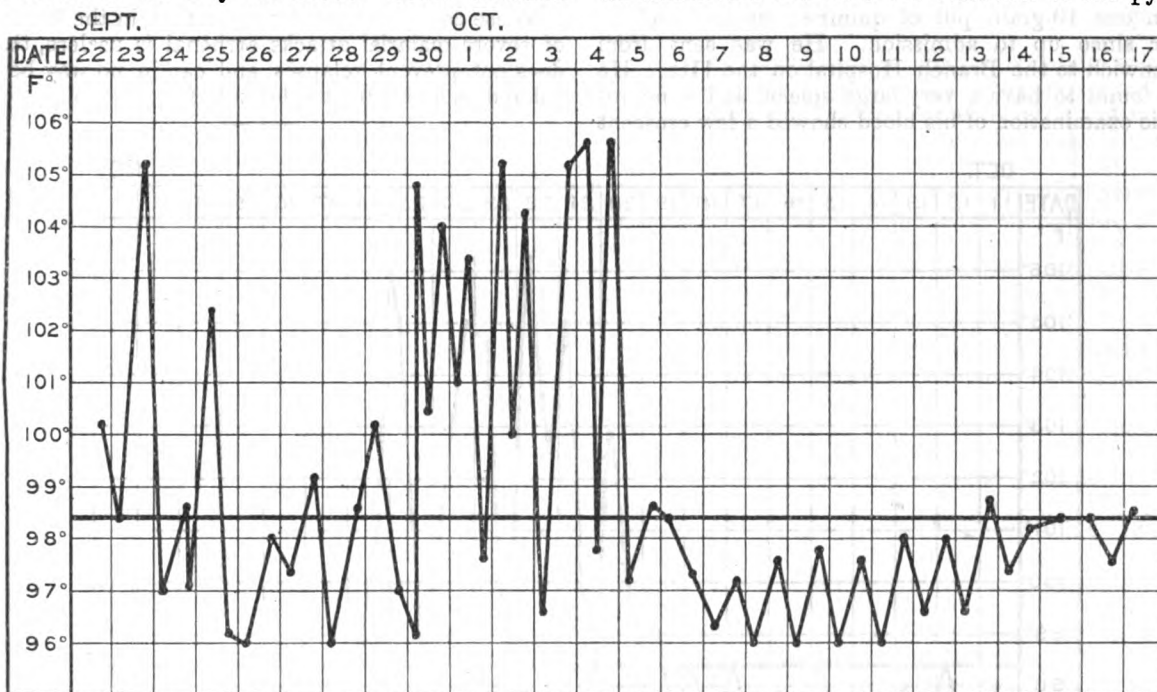
CASE I. ON RE-ADMISSION.

diminished in number. On the 29th the parasites were more numerous and continued to be found in large numbers till October 5th, when quinine was administered. On the 30th he had a slight rigor

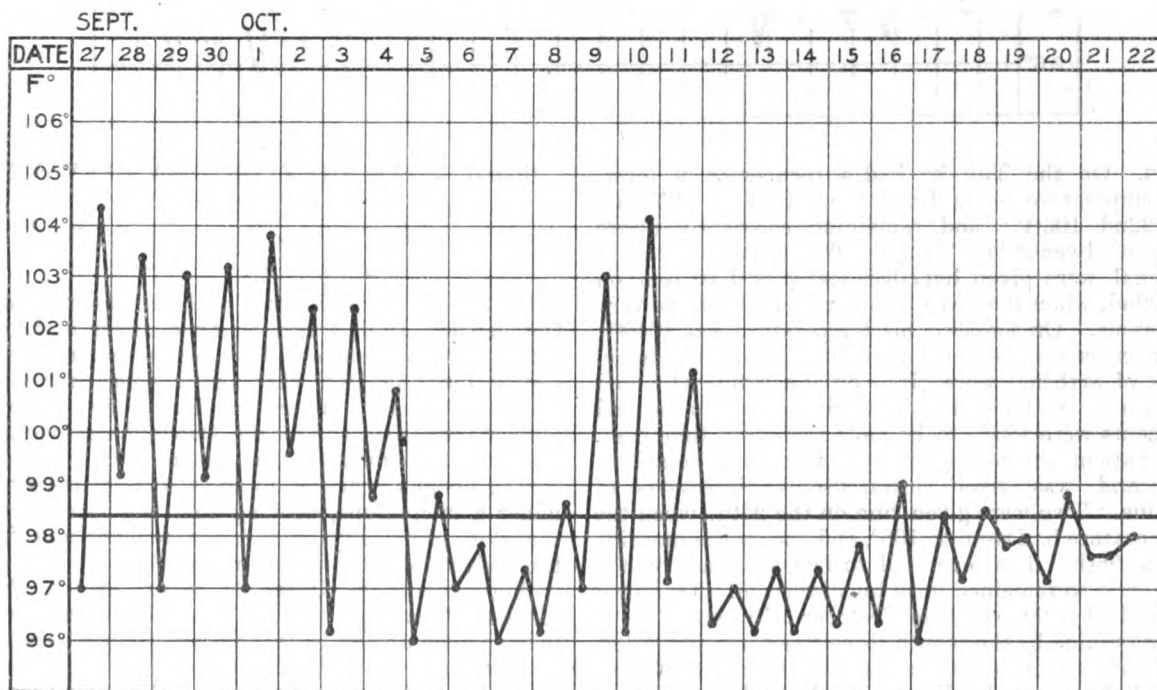
and a temperature of 104.8°. On October 1st, 5 centigrammes of the arrhénal were injected intramuscularly, and continued to be given in this way daily until the 3rd. On the 4th, 10 centimetres were given. The chart temperature showed an irregular rise, usually twice a day, to 105.2° and 105.6°. On the evening of the 4th the patient vomited and passed blood *per rectum*. It was decided to inject quinine at 2 a.m. on the morning of the 5th. At 11 a.m. on the same day the injection was repeated, when the temperature became normal. Five grains of quinine were given by the mouth three times a day afterwards. There were no parasites in his blood on the 6th.

until the 9th, when the patient had a rigor and his temperature rose to 103° . On the 10th he had a rigor and his temperature rose to 104.2° . Ring forms were found to be very numerous. Some crescents

commenced, crescents had been found and were increasing in number each day. Such an increase is the usual accompaniment of natural recovery from an attack and therefore the cessation of the pyrexia



CASE II.



CASE III.

were also found. It was decided to give him injections of quinine; three were administered on the 10th and 11th; the temperature fell to normal on the 12th and so remained until his discharge.

Remarks.—In this case, when the arrhènal was

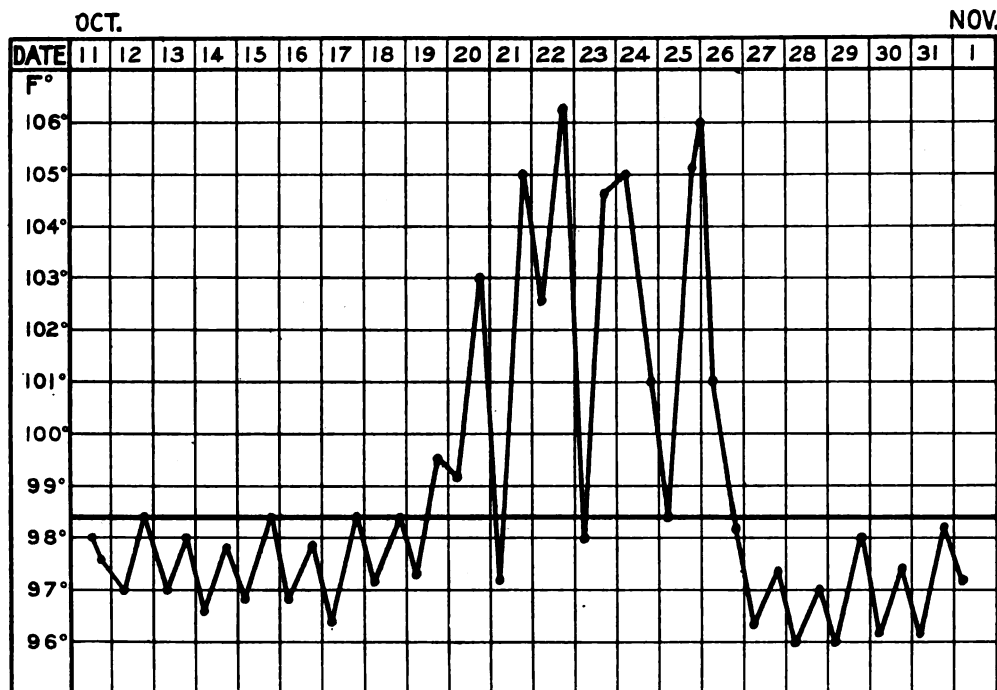
cannot be attributed with certainty to the arrhènal. Though the arrhènal was continued in full doses and for a period of seven days, a severe relapse occurred, which yielded at once to quinine.

CASE IV.—H. J. S., seaman, aged 40. Last voyage

from Mobile, Alabama, U.S.A. Admitted October 11th, 1902. Has had three attacks of malaria, the first being on September 16th. He was admitted to the Seamen's Hospital, Greenwich, on the 9th, and was given one 10-grain pill of quinine; he has had no fever since up to admission. He was sent from Greenwich to the Branch Hospital on the 11th. He was found to have a very large spleen, and a microscopic examination of his blood showed a few crescent

arrhènal were the largest dose recommended by Dr. Gautier, and twice the dose he used in most of his cases. Under quinine both pyrexia and parasites rapidly disappeared.

These cases we consider show that in the treatment of severe malarial attacks arrhènal is useless, that it does not prevent relapses, and can in no way be considered as an efficient substitute for quinine.



CASE IV.

forms. On the 20th he had a recurrence of fever, his temperature being 103.2°; on the 21st 105°; on the 22nd 106.4°; and remaining constantly above 103° for twenty-four hours. Ten centigrammes of arrhènal were given hypodermically at 1.40 p.m. on the 22nd, when the fever forms were found to be very numerous. On the 23rd his temperature was 104.4°; fever forms and crescents very numerous. The injections of arrhènal were given on the 22nd, 23rd and 24th, and at the end of that time fever forms and crescents were found to be more numerous than ever. The patient showed symptoms of cerebral complication and was given intramuscular injections of quinine. Two were given him on the 25th, when the temperature had risen to 106°, and one on the morning of the 26th. The fever fell to below normal on the 26th, and so remained until his discharge. The fever forms had disappeared on October 27th, and he was given quinine by the mouth three times a day. A few crescents remained in his blood. On the 31st they had all disappeared. He was discharged convalescent on November 1st.

Remarks.—Another case yielding readily to quinine, but with a strong tendency to recurrence. No beneficial effects followed treatment with arrhènal. The pyrexia continued uninfluenced by the drug and parasites remained numerous. The injections of

PROOF THAT ANKYLOSTOMA LARVÆ CAN ENTER THE SKIN.

By F. M. SANDWITH, M.D., F.R.C.P.
Professor of Medicine, Cairo.

READERS of this Journal will remember that on October 1st, 1901, I had an opportunity of bringing before English readers Dr. Looss's remarkable discovery, made in 1898, that ankylostoma larvæ could enter the human body by the skin as well as by the alimentary canal; and at the Cheltenham meeting of the British Medical Association in 1901, I was able, by Dr. Looss's courtesy, to show some of his sections under a microscope, proving that the larvæ entered the skin principally by the hair follicles and forced themselves into the subcutaneous tissue.

Since then, Dr. Bentley's paper, in the *British Medical Journal* of January 25th, 1902, has brought independent witness to bear that ankylostoma larvæ in Assam can enter the skin.

Dr. Looss, by three experiments, has now been able to prove the truth of his conviction that ankylostoma larvæ entering a healthy animal by the skin can work their way into the intestine. A puppy had his back gently smeared with a mixture of charcoal and fæces in which ankylostoma larvæ had been bred. Between nine and ten days afterwards the puppy died, and

was found to have anæmia of most of his organs, and plentiful young ankylostoma worms were found in his jejunum. A second puppy was treated in a similar way, and also died on the night between the ninth and tenth days, and showed exactly the same results *post mortem*.

A man who offered himself for experiment was also similarly treated on his forearm, and in his case the first ankylostomum eggs were discovered in his fæces on the seventy-first day, and since then many other eggs have been found in his fæces.

In all three experiments, the fæces of the puppies and the man were regularly examined for some weeks prior to the experiment, in order to be quite certain that they were not already harbouring the ankylostomum worm. Moreover, the worms found in the puppies were all immature, and not one single adult worm was found in their intestines, showing that the infection must have been a very recent one. In Dr. Looss's previous experiments he has found that dogs infested by the mouth also died between the ninth and tenth days, and the ankylostoma worms found in the intestine were in exactly the same stage of development as in the cases now recorded, where the animals were infested by the skin.

Dr. Looss will read a detailed paper upon his experiments at the Medical Congress in Cairo, but has kindly allowed me to send this preliminary notice to readers of this Journal.

REMARKS ON THE INDIVIDUALITY OF "FILARIA DIURNA."

By Louis W. SAMBON, M.D. (Naples).

Lecturer at the London School of Tropical Medicine.

IN the second part of the Report of the Malaria Expedition to Nigeria of the Liverpool School of Tropical Medicine, which is devoted to a compilation of recent knowledge concerning filariasis, Drs. H. E. Annett, J. Everett Dutton and J. H. Elliott suggest the identity of the embryonic filariæ which Manson has called respectively *F. nocturna*¹ and *F. diurna*. Indeed, these gentlemen declare that although many points remain to be cleared up before the question can be settled, yet "the weight of evidence is on the side of the identity of *F. nocturna* and *F. diurna*."

Drs. Annett, Dutton and Elliott base their opinion on the following data, which I quote, as far as possible, in their own words:—

Geographical Distribution.—The distribution of *F. nocturna* corresponds, in certain regions, with that of *F. diurna*, but, as far as is at present known, though there appear to be many lands where *F. nocturna* alone is found, in no district has it been shown that *F. diurna* prevails alone.

Microscopical Appearances of Embryos.—"We were unable to distinguish the embryos in the blood of natives infected with *F. nocturna* and *F. diurna* respectively, by any means whatever."

Numbers of Embryos in Peripheral Blood.—"Here, again, there is a close similarity between the two worms."

Analogy with Avian Filariasis.—"Each species (Avian filariæ) possesses distinct adults, which give rise to a characteristic embryo. This would suggest a similar condition among human filariæ, and thus that *F. diurna* and *F. nocturna*, being indistinguishable in fresh and stained specimens, have a common adult form."

Periodicity.—"Cases of filarial infection occur in whom the hour at which the maximum number of embryos is present in peripheral blood is not midday and midnight, but may be any other hour—3, 6, or 9 a.m. or p.m."

As shown by Mackenzie, Manson and Thorpe, the periodicity depends on the sleeping habits of the definitive host.

The Adult Form.—Manson has suggested that *F. diurna* might be the larva of *F. loa*. *F. diurna* has been found in some cases of natives in which *F. loa* has been removed from the eye, but cases have occurred in which no embryos could be demonstrated in the blood.

The Intermediary Host.—*F. nocturna* has been successfully cultivated in several species of mosquitoes belonging to the genera *Culex* and *Anopheles*. "We were able to cultivate this embryo in *Anopheles costalis*; but all our efforts to cultivate *F. diurna* failed. But this is not remarkable, for if *F. diurna* had been evolved in consequence of the habits of the natives, it is not unnatural to expect that its intermediary host is an insect, probably a mosquito, not essentially nocturnal in its habits, such as *A. costalis*, but one whose habits are diurnal."

Concurring fully with the authors as to the importance of the subject, I propose to discuss the various arguments on which they base their opinion, and, in opposition to their statement, I will endeavour to prove that *F. nocturna* and *F. diurna* represent distinct species, and that *F. diurna* is very probably the larval form of *F. loa*.

Geographical Distribution.—The geographical distribution of *F. nocturna* (*F. bancrofti*) is very extensive. On the other hand, that of *F. diurna* appears to be greatly restricted. Unfortunately, our knowledge of the geographical range of *F. diurna* is as yet very imperfect. *F. diurna* was first discovered by Manson in two patients, one of which came from Old Calabar, the other from the Congo. Later, he found it in the blood of a negress from Dahomey. Recent observations show that it is very common in certain districts of the Lower Niger. Drs. Annett, Dutton and Elliott propose to add the Friendly Islands of the Pacific Ocean to the stations of *F. diurna*, because they believe that the filaria described by Thorpe² "must be taken as *F. diurna*." Their assumption is most gratuitous: Surgeon V. Gunson Thorpe, R.N., during the survey of Tonga by *H.M.S. Penguin*, August—December, 1895, examined the blood of 214 adult Tongans and found a filaria embryo closely resembling the embryo of *F. bancrofti*, but somewhat smaller in size and exhibiting no periodicity. "Ninety-six natives were examined both day and night, and, with two exceptions, all those with filariæ in the blood at night exhibited them in the daytime in equal numbers and *vice versa*." Surgeon Thorpe came to the conclusion that "no sufficient grounds exist for regarding

this filaria as a new species, in spite of the absence of periodicity and certain differences in measurement of its size; and that therefore we must consider it to be *F. nocturna*, the periodicity of which has been altered and modified by the habits of the natives of the Tonga Island." The filaria of Tonga is possibly distinct from *F. bancrofti*, but I fail to see why it must be taken for *F. diurna*.

The fact that *F. diurna* does not prevail in any place alone, while *F. bancrofti* is found in many lands alone, does not in any way prove the identity of these two hæmatozoa, but is merely a proof of the different and wider distribution of *F. bancrofti*.

Drs. Annett, Dutton and Elliott, in speaking of the geographical distribution of *F. bancrofti* and *F. diurna*, make a statement which proves that they consider the term elephantiasis as synonymous with filariasis. They say: "The distribution of elephantiasis (caused by the presence of the adult form of *F. nocturna* in the lymphatic vessels and other sites) is extremely wide." So far as I am aware, it has been suggested, but not proved, that *F. bancrofti* is the cause of elephantiasis. Elephantiasis is a frequent and striking accompaniment of filarial infection, but I think it is more a sequela than a manifestation of the helminthiasis. It is probably due to the bacteria which cause the intercurrent attacks of lymphangitis to which a filariated patient is so liable in the obstructed lymphatic regions of his body. The incidence of elephantiasis within the endemic areas of *F. bancrofti* varies greatly, and a similar condition may be met independently of filariasis.

Microscopical Appearances of the Embryos.—Drs. Annett, Dutton and Elliott state that they were unable to detect any difference between the embryos of *F. diurna* and *F. nocturna*. "They appeared identical in their appearance, characters, measurements and movements in fresh preparations, and correspond in length, breadth, staining reactions, and in the possession of the same number of 'spots,' situated at similar points along the length of the worm, and of the same shape and size. The sheath, a common feature of each, appeared identical."

Having seen only a few badly-stained specimens of *F. diurna*, I am not in a position to make any remark as to the morphological distinctions between *F. diurna* and *F. nocturna*. Dr. Manson states that although practically indistinguishable when seen alive in the blood, *F. diurna* shows a singular difference on dried and stained slides of thickest blood-films. It looks shrunken and thickened and assumes a stiff, rigid attitude, which is perfectly characteristic. In the specimens I have been able to examine, I have certainly noticed this peculiar attitude of *F. diurna*, which is strikingly different to that of *F. nocturna*, usually arranged in smooth, graceful curves. Another feature probably characteristic of *F. diurna* is the recurvation of the tip of its tail, which is present in most specimens. Dr. Manson was the first to notice it, and I drew his attention to an identical recurvation in the embryo of *F. loa*.

Dr. Prout³ found *F. diurna* twice in Sierra Leone. He says: "I am inclined to believe, from measurements of stained specimens (though I am aware this is apt to vary), that *F. diurna* is a slightly smaller worm

than *nocturna*, and that the tail does not taper so sharply." Prout also examined a patient who had had two specimens of *F. loa* removed, one from the loose skin of the penis and the other from the eyelid. He found the peripheral blood swarming with a sharp-tailed embryo. He says the point of the tail was not so sharp as that of *F. nocturna*, but he does not mention whether this embryo had a sheath or whether it observed any kind of periodicity.

Analogy with Avian Filariasis.—Having found easily distinguishable embryos in a few bird filariæ examined by them in West Africa, Drs. Annett, Dutton and Elliott make the remarkable statement that because they are unable to distinguish *F. diurna* and *F. nocturna* in the embryonic stage, these two worms must have a common adult form! A wider study of comparative pathology would no doubt have made them more cautious. As Manson justly observes in his article on *F. diurna* (Davidson's "Hygiene and Diseases of Warm Climates"), "a close anatomical resemblance between two specimens of filaria embryos is no sufficient reason for concluding that they belong to the same species. It is a well-known fact that nearly all filaria embryos closely resemble each other, even although they may belong to widely different species. In many cases the species, as regards their embryos, are only distinguishable by the physiological tests of habit and ultimate developmental change."

Periodicity.—It was chiefly on account of its peculiar diurnal periodicity that Manson described *F. diurna* as the embryo of a distinct species. "I can readily understand," he says, "objection may and probably will be taken by some to the inadequacy of the data from which I infer the existence of a third species of blood-worm in man. My confidence, however, in the regularity of the operations of Nature, even as affecting these lowly organisms, is such, that the one fact of difference of periodicity in the sharp-tailed, sheathed filariæ is to me quite sufficient to prove difference of species. The opposite periodicities of *F. diurna* and *F. nocturna* constitute, from a physiological point of view, a radical and specific difference, seeing that they imply a difference in intermediary host."

Drs. Annett, Dutton and Elliott state that they observed cases in West Africa in whom the hour at which the maximum number of embryos was present in peripheral blood was not midday and midnight, but 3, 6, or 9 a.m. or p.m. But their cases, as they state themselves, were mostly cases of mixed infection, and the overlapping of the two broods with reversed periodicity must be taken into account. Besides, no one has ever stated that the maximum number of embryos of *F. diurna* and *F. nocturna* is found punctually at midday and midnight respectively. Manson, who has discovered this remarkable periodicity, says: "Towards sunset—about 5 or 6 o'clock—they (*F. nocturna*) begin to enter the general circulation. Gradually, as the night wears on, their numbers increase. About midnight they are most numerous. As morning approaches they get fewer and fewer, and by 8 or 9 a.m. they have disappeared. . . . *Filaria diurna* comes into the general circulation about 9 or 10 in the morning, increases in numbers till 1 or 2 p.m., then decreases in numbers, gradually disappearing for the night about 9 or 10 p.m."

Drs. Annett, Dutton and Elliott ascribe the periodicity of *F. diurna* to the merry habits of the West African natives, who "sing and dance the whole night through, especially on moonlight nights!" That the periodicity of *F. nocturna* may be for a time inverted or completely broken up by a change in the sleeping habits of the host, the experiments of Mackenzie and Manson have undoubtedly proved, but I cannot agree with the authors when they state that "such conditions would, in a great measure, account for the variety in the cases of filarial infection we met with in West Africa, and which Thorpe observed in the Friendly Islands, and point strongly to the identity of the two embryos, or rather to the phenomenon of the accommodation of the one or the other, or of an original embryo perhaps exhibiting no periodicity whatever, to the varying habits of the natives who formed their habitat."

Filarial periodicity has not been determined by the sleeping state, as some have conjectured, but is evidently correlated with the life-habits of those insects which have become the liberating agents of the blood filariæ. We can find a large number of similar remarkable adaptations in Nature. Many flowers which open early in the morning are only visited by particular butterflies which leave their nocturnal haunts at the same hour. Other flowers do not open till sunset and they are visited by Hawk-moths, silk-moths, owlet-moths and other *Noctuæ*, which commence their ramblings when dusk sets in. Then, again, the development of flower-scent is simultaneous with the time of flying of certain insects. The flowers of *Hesperis tristis* and other flowers which are visited by small nocturnal moths give off no scent during the day, but exhale a strong hyacinth odour at twilight; on the other hand, many flowers visited during the day become scentless at night. Although a change in the sleeping habits may bring about an inversion of periodicity in cases of *F. nocturna*, just as the application of a wet sponge on a limb harbouring the adult *F. medinensis* may induce this worm to eject some of its embryos inopportunely, I believe; he swarming of *F. diurna* in the peripheral blood during daytime is not due to the wakes of the natives of West Africa, but to the diurnal habits of the intermediary host of *F. diurna*.

It is to be regretted, I think, that Drs. Annett, Dutton and Elliott, who ascribe the periodicity of *F. diurna* to the midday naps of the Kroo boys, did not make any experiments to find out whether they could invert the periodicity of *F. diurna* by causing the natives to sleep regularly during the night.

In one case of *F. diurna*, most carefully investigated by Dr. Manson, the patient kept very regular habits and slept only at night, yet the diurnal periodicity of his filaria embryos was constantly the same.

The Adult Form.—Dr. Manson has suggested that *F. diurna* may be the embryo of *F. loa*. He says: "The patient who supplied me with blood for my observations on *F. diurna* informed me that when a child he had a *F. loa* in his eye; that after a time it disappeared spontaneously; and that this is not an uncommon parasite in his country." Other cases of the simultaneous presence of *F. loa* and *F. diurna* have been mentioned by Drs. Prout, Annett, Dutton and Elliott, but the latter three gentlemen look upon

it merely as an ordinary coincidence, and oppose the idea of any possible connection by stating that cases of *F. loa* have occurred in which no embryos could be demonstrated in the blood. I believe Manson's suggestion deserves great consideration. Several filariæ have been mentioned as parasitic of man on the West Coast of Africa. We know of the presence of *F. bancrofti*, *F. perstans*, *F. medinensis* and *F. volvulus*. Of these filariæ we know both the early larval forms and the mature adult forms. *F. bancrofti* and *F. perstans* have a wide distribution in Africa and in other continents; *F. medinensis* is also very widely distributed, but it is absent in many parts of West Africa. *F. volvulus* is apparently limited to West Africa. Dr. Prout has recently described a very large, non-sheathed, blunt-tailed filaria embryo in the blood of a native of Sierra Leone. This filaria embryo, which he has called *F. gigas*, has evidently no connection whatever with *F. loa*. There remain, therefore, only a filaria embryo, *F. diurna*, and an adult filaria, *F. loa*, both very common in the natives of West Africa and both with exactly the same geographical distribution, a distribution singularly limited to West Africa. These coincidences strongly suggest a connection between *F. diurna* and *F. loa*.

But, if *F. diurna* be the embryonic form of *F. loa*, how shall we explain the fact that embryonic filariæ are not always found in patients known to harbour *F. loa*? I think I can explain this fact quite satisfactorily. *F. loa* develops very slowly within the body of its definitive host; it may not reach maturity for many years, as proved by the cases described by Mitchell,⁴ Ludwig and Saemisch.⁵ In its mature stage, and long before attaining maturity, it may frequently appear about the eyes, cruise round the eyeball in the cellular tissue between the conjunctiva and sclerotic, or pass rapidly from one eye to the other through the cellular tissue under the skin at the root of the nose. Occasionally, it may be felt wriggling under the skin of the forearm and fingers. The slow development of *F. loa* and the frequent excursions of this parasite during development explain the great differences in the size and development of the worms removed from the conjunctiva of various patients. It is evident, therefore, that a patient may be suffering from *F. loa* for years before any embryos can appear in his blood. Being usually obnoxious during its restless developmental stage, the *loa* is frequently removed before it has attained full maturity. The natives use for this purpose a bamboo spike. Then, again, the patient may harbour only one *loa* producing too small a number of embryos for diagnosis, or he may have only a male *loa*. Another interesting and most suggestive coincidence which has not been noticed hitherto, is that while children are very frequently the bearers of *F. loa*, only adults appear to be infected with *F. diurna*. Drs. Annett, Dutton and Elliott state that "In the examination for malarial parasites of blood specimens from a large number of native children of all ages up to about 18 years, we encountered a single filarial embryo only in one case (specimens taken during the day were examined only), aged 11, out of 390 cases. In view of the number of adults infected with *F. diurna* in the same districts this is remarkable, and further tends to support the idea that the extent of infection increases during the period of childhood,

until, when adult age is reached, there are a sufficient number of mature female filariæ in the body to give an observable number of embryos in peripheral blood during the usual examination for microscopical purposes."

Probably *Filaria loa*, like *F. equina*, after attaining full maturity lodges itself in the peritoneum or in the pleura and there attends to parturition. Prior to reaching full maturity, *F. equina* is frequently found in the eye of its definitive host, which may be a horse, an ass, or a mule. Professor Blanchard⁶ has recently pointed out that *F. loa* exhibits at its cephalic extremity two papillæ in the form of spinules. Similar structures are seen in *F. equina* and other filariæ of various animals. *F. labiato-papillosa* of cattle and deer is another filaria of the serous membranes which, before attaining full maturity, may be found in the eye of its host. The embryos of both *F. equina* and *F. labiato-papillosa* are found in the peripheral blood of their definitive hosts; it is therefore quite reasonable to infer by analogy that the embryos of *F. loa* should be found in the peripheral blood of man.

The female specimen of *F. loa* removed from the eye in Dr. Robertson's⁷ case was almost mature, and contained fully-developed embryos in the lower part of its uterine tubes. Dr. Manson who examined this specimen says: "The more mature embryos resemble in size and shape those of *F. nocturna* and *F. diurna*, but in consequence of the method of mounting it is impossible to say if they are possessed of a sheath or not. If they are possessed of a sheath, I should say that they are practically indistinguishable from the parasites mentioned."

From the description and drawings of the embryos of *F. loa* in the case described by Ludwig and Saemisch, I am led to believe that the development of the embryo in *F. loa* is probably the same as in *F. nocturna*, and that in both cases the embryo stretches its egg-membrane into a "sheath."

From the above facts, I think we may conclude that the connection between *F. diurna* and *F. loa* is very probable, whilst the identity of *F. diurna* and *F. nocturna*, suggested by Drs. Annett, Dutton and Elliott, has no foundation whatever.

REFERENCES.

¹ Of some parasitic filariæ only the larval form is known, of others only the adult form. This way of finding the worm separately in one or other stage of development has given rise to an inappropriate and confusing nomenclature. Each parasite has received two different names. This is by no means the only instance of the kind in the nomenclature of natural history. A well-known example is that of the Mexican salamander. It was called *Arolotl* when found in water, still retaining its gills, and *Amblystoma* when found on land without gills and with other slight modifications. The two forms were originally referred to different genera, and, indeed, it was only in 1865 that it was discovered that *Arolotles*, though able to retain the larval condition permanently, might, under suitable conditions, assume the salamander stage and become *Amblystomas*. *F. nocturna* should be called *F. bancrofti* after the name of its parent form discovered by Bancroft.

² V. G. THORPE, "*Filaria Sanguinis Hominis* in the South Sea Islands." *British Med. Journ.*, 1896.

³ W. T. PROUT, "Filariasis in Sierra Leone." *British Med. Journ.*, Sept. 20th, 1902.

⁴ H. MITCHELL, "Report of a Case of a Guinea-Worm in the Eye." *Lancet*, ii., p. 533, 1859.

⁵ H. LUDWIG and TH. SAEMISCH, "Ueber *Filaria loa* im Auge des Menschen." *Zeitschrift für wiss. Zoologie*, lx., pp. 726-743, 1895.

⁶ R. BLANCHARD, "Nouveau cas de *Filaria loa*." *Archives de Parasitologie*, ii., No. 4, p. 504, 1899.

⁷ D. A. ROBERTSON, "Case of *Filaria loa*." *Transactions of the Ophthalmological Society*, xv., 1895.

(To be continued.)

REPORT OF A CASE OF BILHARZIA FROM THE WEST INDIES.

By P. MANSON, C.M.G., M.D., F.R.S., LL.D. (Aberd.).

With the exception of Mesopotamia, Cyprus and Mauritius, bilharzia disease has hitherto been supposed to be peculiar to Africa. The following case shows that the parasite has a wider range. Personally, until I came across this case, I never encountered the disease in patients from the West Indies, but its occurrence in a white man from that part of the world practically proves that in certain of these islands, if not in all, it must be by no means uncommon amongst the coloured population. Now that attention has been directed to the subject, we may hear from time to time of similar cases. It is evident that the distribution of this and similar parasitic diseases depends on the presence or absence of the efficient intermediaries. Possibly our zoologists may be able to point to some mollusc or arthropod which the West Indies and Africa have in common, and thereby indicate the long-sought-for, but hitherto undiscovered intermediary host of *Bilharzia hæmatobia*. Another African disease, guinea-worm, was at one time said to be endemic in Curaçao and others of the West Islands, and in a limited area in Brazil. I understand the disease has disappeared from Curaçao, and we have no longer accounts of its presence in the Brazils. It may be that bilharzia has obtained a similar precarious footing in the Western Hemisphere, and that subsequently it will disappear from that part of the world; meanwhile we can assert its presence there as a fact.

I may mention that the patient, an Englishman and a professional man, came to me as a private case complaining of vague symptoms, lumbar pain, headache, &c. As I could not account for the symptoms, and as he looked anæmic, and knowing that he came from a place where *ankylostomum duodenale* is very prevalent, the idea that he suffered from ankylostomiasis occurred to me. I made an examination of his feces and so discovered the ova of bilharzia. In this case, as so often happens in bilharzia ova from the alimentary canal, the spine is placed laterally.

I am indebted for the following notes on the case to Dr. Ross and Dr. Daniels, of the London School of Tropical Medicine.

Previous History.—The patient is an Englishman aged 38. Five years ago, in Antigua, after a heavy day's work he felt a dull pain in the lumbar region; it went off after rest, but would come on again after active exercise. During the last year this pain has increased in severity and duration; was invalided home on account of it. Has resided in the West Indies chiefly for fifteen years, and has had many attacks of malaria. Never passed blood in his urine; never noticed blood in his stools.

Present State.—Temperature 97°; tongue slightly coated; is slightly anæmic; complains of right frontal headache; has some enlargement of liver and spleen.



TUMOUR OF THE NOSE.

Illustrating the Article by J. NUMA RAT, Medical Officer, Leeward Islands, West Indies.

Microscopic Examination.—*Fæces*, bilharzia ova not numerous; generally distributed throughout the fæcal mass; lateral spined. *Urine*, no ova or blood-cells.

Blood.—(By G. Duncan Whyte.) Hæmoglobin, 84 per cent.; red blood-cells, 4,650,000; white blood corpuscles, 8,200; polymorphonuclear leucocytes, 49 per cent.; lymphocytes 21 per cent.; mononuclear leucocytes, 17 per cent.; eosinophiles, 12 per cent.; intermediate, 1 per cent.

History of Patient's Residence in West Indies.—By himself. I went from England to Antigua in May, 1887. In the place I lived in there are a good number of swamps, but my house was a mile from the nearest; water was obtained from cisterns (iron) and ponds, was often stale, and contained visible living things; had two or three attacks of malarial fever, one severe.

I removed to Anguilla in 1889. It is a flat and dry island. The drinking water came from stone cisterns, the bath water from wells. My health here was fairly good.

Moved to St. Kitts in 1891. This is a mountainous island, no swamps within six miles of my residence; water from public service reservoir conveyed in pipes. My health was fair, but had two or three "run-downs" and one or two attacks of fever. Came to England in June, 1894, and returned to St. Kitts in January, 1895.

In September, 1896, I returned to Antigua—same district, but nearer to swamps; water from stone cistern; plenty of frogs, &c., in water, which was usually boiled; health very unsatisfactory; pain in back severe, but generally yielding to rest and treatment.

In June, 1898, I came to England and returned in January, 1899. In December, 1900, I returned to St. Kitts, but to a different neighbourhood; house 600 feet above the level of the sea; nearest swamp three miles; drinking-water from private mountain source; water for baths, &c., from open mountain source (very unsatisfactory at times), passing through a village and used by everybody. On two or three occasions I got an "itch" in the bath called locally "cow-itch." My health, at first good soon failed, and for the whole of this year I have had pain in the back and headache on right side and right eye; also pains in the knee-joints and always tired. Was invalided home in July.

I ought to have added that between 1891 and 1901, I have been to Nevis on short visits many times; water there is good. To Montserrat two or three times; water there also good. I spent five days in St. Thomas in 1894, and five weeks in Barbadoes in January, 1900. I have never been to Africa or anywhere else except the British Islands.

TUMOUR OF THE NOSE.

By J. NUMA RAT.

Medical Officer, Leeward Islands, West Indies.

I SEND the photograph of a young negro who consulted me about seven years ago, in St. Christopher, West Indies, about the tumour on his nose represented in his likeness. The growth was uniformly

distributed over the tip and sides of the nose, and was covered by a thick, rough, yellow crust, which was firmly adherent to the subjacent tissues. The colleagues to whom I sent the patient for examination concluded, chiefly from the presence of enlarged glands, that the case was one of syphilitic chancre; but I failed to detect any other symptoms corroborative of this diagnosis. The patient did not remain sufficiently long under my care to allow me to note the progress of the case. It will be observed on comparing the photograph with the illustrative likenesses printed with Dr. Mitchell's article in the JOURNAL OF TROPICAL MEDICINE of May 1st, 1902, that there is a great resemblance between the tumour on the man's nose and that shown in the case of the girl whose picture is on the right hand side of the illustration.

St. Kitts, West Indies,

Nov. 10th, 1902.

FILARIASIS AND SLEEPING SICKNESS.

By J. NUMA RAT.

Medical Officer, Leeward Islands, West Indies.

THE correspondence which has lately appeared with reference to the relation of filariasis to sleeping sickness, has reminded me of a case in which I performed a thigh amputation in a man suffering from elephantiasis of the foot and the leg below the knee. Out of fifteen amputations of the leg for various diseases performed by me at the Roseau Infirmary, Dominica, West Indies, during fifteen months, his was the only case which ended fatally. The wound was slow in healing but was quite healthy when, about the third week after the operation, the patient began to fall asleep at most unexpected moments. He would fall asleep while eating, during micturition, &c., &c. He would wake on being spoken to, smile, eat a little, and fall asleep again, and it was necessary to keep shaking him to keep him awake so that he might be fed. He continued in this state until his death, never having exhibited any other symptom of disease.

St. Kitts, West Indies,

Nov. 10th, 1902.

THE BACTERIA OF THE MOUTH AND THE ANTISEPTIC PROPERTIES OF ODOL.

By PROFESSOR STANLEY KENT, M.A.

Director of the Clinical and Bacteriological Research Laboratory of the University College, Bristol.

ONE of the most noteworthy advances to be observed at the end of the nineteenth century is the rapid spread of education amongst all classes of the population. It is an age of education, and this is not less true of matters which concern the individual than of matters which concern the State.

Notably in matters of personal hygiene there has been a great change taking place amongst the common people, and to-day the necessity for a daily bath is recognised by many who would have scorned such a suggestion only a few years ago, whilst in other directions also, a knowledge of physiology is leading to a more rational mode of life.

There is one department of personal hygiene, however, in which but little progress has yet been made. We refer to

the hygiene of the mouth, and it is almost certain that the lack of advance in this particular is due to ignorance of the inconvenience which a neglect of the mouth involves. Unfortunately, scarcely anyone realises that it is to such neglect that decay of the teeth is largely due—decay which is a source of trouble to almost everyone at some period of their lives. And hence this neglect continues.

It is true that most people in the higher walks of life appear to recognise the desirability of cleaning the teeth. But what does "cleaning the teeth" really mean in their case? It means a perfunctory brush round in the morning, with or without the use of tooth-powder, whilst in the great majority of cases no further attention is given to the cleansing of the mouth in the whole of the twenty-four hours.

The result of such a state of things is easily imagined, provided that one is acquainted with the conditions that obtain in the mouth.

In the first place it may be said that the matutinal brushing of the teeth, though a pleasant exercise, is really not of much practical use, simply because the first meal of the day follows almost immediately afterwards, and the mastication of food, with the accompanying flow of saliva, would of itself, without any extraneous aid, cleanse the mouth very effectually from the accumulated secretions of the past night. But on the other hand, the taking of food, in most cases, results in the leaving in the various small cavities at the bases of the teeth, between the teeth, and in other tiny receptacles, remnants of food which escape the action of the tongue, and lurk for an indefinite time in the mouth. This, then, is the first term in the series—the lodging in small cavities in the mouth of remnants of food.

The second stage is the formation, by various fermentative processes, of acid substances from these food particles. Fermentation takes place in the mouth partly through the action of the salivary ferment, whereby the starchy material of the food is converted into sugar, and partly through the action of micro-organisms, whereby the sugar already formed is further changed into various acids. It is this acid which produces great injury to the teeth, and lays them open to the attacks of other micro-organisms, with the result that decay sets in, the teeth are destroyed, and all the evils of disturbed digestion follow. It is on this account that starchy food is more harmful to the teeth, if neglected, than sugar, often given in the form of sweets to children, for whilst the sugar is rapidly dissolved and washed away by the saliva, the starchy material, adhering to the teeth, is converted gradually into sugar, and then into acid, and consequently acts for a prolonged period of time, and produces a more profound effect.

In order to show how greatly the teeth may be damaged by neglect, it is only necessary to make an experiment in which the conditions existing in the (uncleaned) mouth are copied as closely as possible. If, for instance, we take a mixture of bread, meat, sugar, &c., and rub it up with saliva, and afterwards immerse freshly drawn sound teeth in the mixture, we find in the course of a very few days that a striking change has occurred. The teeth are no longer hard as they were in the fresh condition. Their hardness, upon which they depend for their usefulness in the mouth, has almost entirely disappeared, and has been exchanged for a softness so pronounced that a needle can be passed through their substance with ease. The teeth have, in fact, lost their ordinary characteristics, and are no longer at all adapted for the mastication of food.

If we allow the process to go further it is not difficult to show that soon other changes, almost exactly similar to those occurring in a decayed tooth in the mouth, take place, whereby the tissues of the teeth are gradually destroyed. If, then, this change can be produced so easily outside the mouth, it will be produced even more easily when the teeth are *in situ*, because the warmth of the body will favour the changes, and hasten the action. Evidently, then, one of the first things to be considered in arranging a scheme for the preservation of the teeth is the complete removal of remnants of food from the mouth, and it is abundantly evident that a perfunctory brushing of the teeth once a day, and that once in the morning, immediately on rising, is altogether insufficient for the purpose.

nants of food from the mouth, and it is abundantly evident that a perfunctory brushing of the teeth once a day, and that once in the morning, immediately on rising, is altogether insufficient for the purpose.

It is, indeed, necessary that the teeth should be cleansed carefully and thoroughly *after every meal*, and difficult and perhaps tiresome as this will appear to those unaccustomed to the discipline, there yet can be no doubt that it is the only way, and compared with the agony of toothache, the discomfort of ill-health from indigestion, and the irritation of having a large dentist's bill to pay, there can be no doubt that it is, after all, a small price to pay for perfect teeth and a healthy mouth. More especially is it necessary to ensure a thorough cleansing of the mouth after the last meal of the day, or immediately before retiring to rest. For if this is neglected, and one retires with food particles still in contact with the teeth, during the whole of the night the fermentative changes, with consequent damage to the structure of the teeth, will be in progress.

A remarkable illustration of this is afforded by the condition often observed in the teeth of the children of the poor, amongst whom it is common habit to give to the child, when in bed at night, biscuits or other starchy food, with the object of quieting it. The result is that the child falls asleep whilst still sucking the biscuit, the teeth are left all through the night covered with remnants of starchy material, which undergoes fermentation, and produces untold mischief.

As pointed out by Mr. Denison Pedley, in his valuable book, "The Hygiene of the Mouth," "the habit of giving bread or other starchy food to a child when in bed at night should be entirely prohibited."

It is evident, then, to anyone who will give the least attention to the matter, that it is of prime importance to cleanse the teeth frequently, and especially to cleanse them thoroughly the last thing at night. But this is not all. As mentioned above, it is to bacteria that the ultimate decay of the teeth is due, the action of these parasitic plants commencing as soon as the fermentative products of the food remnants have sufficiently softened the tooth substance to allow of their invading the tissues. And we may say at once that it is altogether impossible to get rid of these countless multitudes of parasites by any amount of brushing once, twice, or a hundred times a day. They are far too small to be affected by any such treatment, they lie in crevices far too tiny to be reached by bristles, be they ever so fine, and they adhere to the surface of the tongue and cheeks in such a way as to defy the most energetic brushing.

What, then, is to be done? Obviously, just as we use a brush for the removal of the food particles, so we must use something equally efficacious for the removal of the bacteria. We must, in short, use antiseptics, *i.e.*, substances which will act as poisons to the bacteria, and so destroy them. But it is clear that whilst acting as poisons to the bacteria, these substances must be harmless as far as the mouth is concerned, else we may be in a worse plight than ever. And the great difficulty has always been to find a substance which, whilst being sufficiently active in killing off the bacteria of the mouth, should at the same time be harmless to the tissues of the person using it.

Long and elaborate series of experiments on this subject have been carried out by continental bacteriologists, most of the well-known antiseptics having been thoroughly tested. Up to comparatively recent times, however, no single substance could be said to possess all the qualities desired, for whilst some were indeed powerful antiseptics, they produced injury to the lining membrane of the mouth, others possessed insufficient antiseptic power from the first, whilst others, again, whilst being satisfactory at first, soon lost their activity when stored, or sent out in the ordinary course of trade.

And finally, many of the best antiseptics possess so nauseous a taste that their daily use would be practically out of the question.

Recently, however, there has appeared on the market a substance which, whilst possessing a powerful antiseptic action, yet is pleasant to the taste, is permanent in solution, and is altogether harmless to the lining membrane of the mouth; so that here, at last, we appear to have found an ideal substance for the cleansing of the mouth, a substance which may be used daily, which will do no harm, which is pleasant enough for children to use it readily, and which may be depended upon to do its work thoroughly.

The substance appears under the trade name of *Odol*, and is to be obtained in the form of a refractive liquid of a pleasant odour and aromatic taste, which forms a milky liquid when mixed with water. The reaction of the liquid is neutral, an essential point, as an acid would bring about damage to the teeth similar to that already described as due to fermenting food particles in the mouth, whilst any trace of alkalinity would damage to a greater or less extent the lining membrane of the mouth.

We see, then, that the preliminary requirements of a neutral, pleasant-tasting liquid are satisfied. It remains to ascertain whether the substance under consideration has that antiseptic action, and is as efficacious a poison to the bacteria as is desirable. For the purpose of ascertaining the truth on this point, a series of experiments has been made, the results of which are briefly given below.

The first set of experiments was made with the view of ascertaining the action exerted by the solution of *odol* in various strengths upon bacteria when grown in tubes in the ordinary manner in a pure condition. And we may say at once that we were at first somewhat disappointed with the results.

The first organism selected for experiment was the diphtheria bacillus, as this is frequently present in the mouth, and it is desirable that when present it should be destroyed, as, although it may produce no symptoms in the person whose mouth it inhabits, it may yet be a source of infection to numberless other individuals, who may in consequence suffer from the disease in a severe form.

The experiments consisted in taking a tube containing a pure growth of the diphtheria organism, pouring into the tube a sufficient quantity of the diluted *odol* solution to completely cover the growth, leaving it in contact with the organism for a period of time varying from a minute to half an hour, then pouring off the solution and thoroughly washing away all traces of the antiseptic by means of successive changes of sterile water. Afterwards, some of the original growth was transferred to a new culture medium, and the tube carefully watched for signs of growth. In nearly every experiment, at the end of a couple of days, a distinct growth had taken place, thus showing that the original culture had not been killed by the treatment. It remained then to test the solution under the exact conditions existing in the mouth, since, after all, results obtained under other conditions are of comparatively small value so far as the merit of the solution as a mouth-wash is concerned.

For this purpose it was necessary to select a different organism, because it is inadvisable to place the diphtheria bacillus in the mouth, and when it is found occurring in the mouth in the case of a patient, it is necessary to get rid of it as quickly as possible without reference to experiment. The organisms selected were those usually associated with disease of the teeth, for it is these especially which are concerned, and which inhabit the mouth normally, and which, therefore, the solution will be used for destroying in ordinary practice.

The experiments followed closely upon those of Dr. Carl Roese, who has already tested a great number of fluids of different composition. The mouth was first of all washed out with water which had been freed from all living organisms by prolonged boiling. After this preliminary washing had been accomplished, a fresh quantity of sterile water or broth was taken, and the mouth washed out with it for a definite time, usually one minute. At the end of this period the fluid was collected in a sterile dish, and carefully measured.

A minute measured quantity of the fluid was then taken and thoroughly mixed with sterile jelly, which was subsequently spread over the surface of a glass plate. The plate was then placed in an incubator kept at the temperature of the body, and examined at definite intervals for evidence of growth.

As the jelly set, each organism present was fixed in the position it happened to occupy at the moment, so that any further development necessarily took place in that exact spot. Favoured by the warmth, each germ speedily increased and multiplied, until it produced a colony visible to the naked eye. By counting these colonies, some idea of the number of organisms originally present in the added wash water was formed, and by counting the colonies in a number of plates and taking the average, a normal standard was obtained with which to compare the numbers obtained after the action of the fluid to be examined.

After the above experiment had been carried out, the mouth was washed with the solution to be tested, in the present case with *odol* of various strengths, which was allowed to remain in contact with the mouth lining for periods which varied, but which were usually one minute.

Then the mouth was washed out thoroughly with sterile water, and afterwards again washed with sterile water or broth as above, the washings collected and measured, and plates of jelly prepared as in the former test. The plates were placed in the incubator at the temperature of the body, and examined from day to day in order to ascertain whether any development of the contained organisms had taken place. The experiments were repeated again and again, so as to obtain an average from which a just estimate of the results might be formed.

The general results are expressed in the table attached, in which the values are calculated as percentages.

Taking the first experiment, at the end of three days, there were in the plate prepared from the wash water taken before treatment, 400 colonies, each of which must have sprung from at least one original organism in the mouth. The amount of water used for making the plate was one drop, whilst the total quantity used for washing the mouth was 105 drops. Multiplying the 400 colonies contained in one drop by the 105 drops used, we obtain 42,000 as the number of organisms capable of growth in the wash water taken before treatment.

Having thus obtained a number representing approximately the normal average of organisms present in the mouth, a second set of experiments was made as follows: The mouth was washed out for one minute with *odol* solutions of various strengths. It was subsequently washed out with sterile water, and afterwards sterile water or broth was used to wash it out once more. From the last washing, plates of jelly were made as in the former experiments, and these plates were placed in the incubator at the temperature of the human body. They were carefully examined from day to day for evidences of growth. The results obtained varied with the strength of *odol* employed. With the stronger solutions, very few colonies made their appearance, thus showing that the action of the antiseptic had been so complete as to leave scarcely any germs capable of growth in the wash water used for preparing the plate.

Comparing this with the result obtained before *odol* had been used, we see that in the one instance 42,000 organisms were taken up by the wash water from the mouth, whilst in the other scarcely a living organism was left. Such a result must be regarded as being exceedingly satisfactory, for it proves that *odol*, which we have before seen to possess pleasant taste and smell, and to exercise no harmful action upon the teeth or mouth lining, yet when brought into contact with the mucous membrane is capable of exerting such a powerful antiseptic action that practically every organism brought into contact with it is destroyed.

It remains to explain the apparent anomaly of the solution failing to kill the diphtheria organisms when growing as a pure culture in a tube, and yet being able to clear the mouth of organisms when used as a wash.

The explanation of this is to be found in the fact (already pointed out by Reese and Frey, and more particularly in an exhaustive study of the subject by Hefelmann), that the antiseptic of odol is of itself indifferent, and must first be broken up into its antiseptically effective elements before it can develop its antiseptic power. This breaking up is effected in the test-tubes by lyes, but in the mouth by the action of the saliva, and by the septic germs themselves, but most of all by the action of the mucous membrane. This last action is of particular importance because, in consequence of the absorption of the odol mixture by the mucous membrane, large quantities of the antiseptic are mechanically deposited on the mucous membrane, and cover it with a layer. The products of the breaking up of the substance are phenol and salicylic acid, whose antiseptic properties are well known. Injurious effects are, however, completely precluded, inasmuch as these two substances are produced only in such quantities as result from the breaking up by the saliva, &c., whilst the rest, as the investigations of the above-named authors show, is changed into harmless compounds, probably with the albuminous substances which are present.

That this result is actually achieved in practice has already been shown.

As the result of these experiments we arrive at the conclusion that in the antiseptic mouth-wash known as odol we have a medium which is neutral, without action either on the teeth on the one hand, or on the mucous membrane of the mouth on the other, which has an agreeable taste and smell, and which yet possesses antiseptic powers amply sufficient, when properly used, to render innocuous any organisms found in the mouth, and so to protect the teeth from decay and the gums from disease.

From a daily use of this solution nothing but good can result, and there could perhaps be no better means of ensuring sound teeth, and consequently, comparative freedom from digestive troubles to an advanced age, than to insist on a daily, and especially a *nightly*, use of odol as a mouth-wash, combined with an efficient use of the tooth-brush for the clearing away of the grosser particles adhering to the teeth.

TABLE SHOWING EFFECT OF ODOL IN VARIOUS STRENGTHS IN DESTROYING OR INHIBITING ORGANISMS IN THE MOUTH.

| Percentage of Odol Solution employed | PERCENTAGE OF COLONIES IN EXPERIMENTAL PLATES (Average) | | Percentage of Organisms Killed or Inhibited by Treatment |
|--------------------------------------|---|---------------|--|
| | Untreated Mouth | Treated Mouth | |
| Per cent. | Per cent. | Per cent. | Per cent. |
| 25 | 100 | 0 | 100 |
| 20 | 100 | 5 | 95 |
| 15 | 100 | 20 | 80 |
| 10 | 100 | 55 | 45 |
| 7 | 100 | 60 | 40 |
| 5 | 100 | 66 | 33 |
| 2 | 100 | 76 | 24 |
| 1 | 100 | 82 | 18 |

In the above table, fractions smaller than 1 have been neglected.

ANKYLOSTOMIASIS IN ENGLISH MINES.—At the Dolwath Mine, Camborne, Cornwall, it is found that a large portion of the men are suffering from ankylostomiasis. The Home Office, some six weeks ago, instituted an enquiry into the ventilation of Cornish mines and the prevalence of miners' phthisis, and it appears that Dr. J. S. Haldane, F.R.S., during the course of the enquiry, discovered the presence of ankylostomiasis amongst the more anæmic miners.

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THE

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RECORDS OF THE EGYPTIAN GOVERNMENT SCHOOL OF MEDICINE.

IN view of the approaching Medical Congress in Cairo, it behoves us to draw attention to the excellent work being carried on by the eminent men attached to the Kasr-el-Ainy Hospital and School in Cairo. The staff of the Egyptian Government School of Medicine, under the presidency of H. E. Ibrahim Pasha Hassan, and the directorship of H. P. Keatinge, M.B., includes such well-known names as those of Dr. G. Elliot Smith (Professor of Anatomy) ; W. A. Schmidt (Chemistry) ; Dr. Nolan, LL.B. (Forensic Medicine) ; H. Bitter (Hygiene) ; Dr. F. M. Sandwith, F.R.C.P. (Medicine) ; Dr. P. C. S. Tribe (Clinical Medicine) ; Dr. E. C. Fischer (Ophthalmology) ; Dr. W. St. C. Symmers (Pathology) ; O. Dinkler (Pharmacology) ; Dr. W. H. Wilson, M.A. (Physiology) ; Mr. F. C. Madden, F.R.C.S. (Surgery) ; Mr. Frank Milton (Clinical Surgery) ; Dr. A. Looss (Helminthology).

The records of the School recently to hand consist of a large volume, beautifully illustrated and replete with interest.

Professor Sandwith contributes an article on "The History of Kasr-el-Ainy from the year A.D. 1466 to 1901," but in his introduction to the subject traces "the earliest known triumphs of the healing art" in Egypt to a period several thousands of years before the Christian era. The first mentioned physician in history is Sekheten-anc, chief physician to a king of the fifth dynasty who lived about B.C. 3400, and "during most of the Egyptian dynasties physicians taught with the priests in the temple schools." The oldest medical book now existing is the Ebers papyrus, which "was written about B.C. 1550, though much of its contents are of far greater antiquity." The stumbling block to dissection seems to have been the belief that the soul was wounded if the body was dissected, yet specialists for the eyes, head, teeth and internal organs are mentioned by Herodotus in the fifth century B.C.

Human dissection, Professor Sandwith relates, was allowed for the first time for a few years in the Alexandrian School under Ptolemy I., who founded the Museum and Serapeum with their large libraries about B.C. 300.

From 750-1250 A.D., Arabic writers represented the highest form of medicine, and a literary Arabic language was formed and spread over nearly half the then known world.

The first hospital in Egypt was built in A.D. 875; and lunatic asylums existed in Egypt long before they were known in Europe, of which the Muristan, built in 1279, served as the lunatic asylum of Cairo until about 1856. The Kasr-el-Ainy, originally a palace (Kasr), built by Ibn-el-Ainy, played an important part in military history, and during the invasion of Egypt by Napoleon was converted into a military hospital. Subsequently it was utilised as a barracks and a school, and it was not until 1837 that Clot Bey succeeded in establishing a hospital and medical school at Kasr-el-Ainy.

The present school is highly creditable to the Egyptian Government and to the staff who carry on the work.

There is now almost every facility provided for the staff and others to try and elucidate some of the unsolved problems of tropical pathology, such as malaria, dengue, dysentery, liver-abscess, leprosy, tetanus, Oriental sores, pinta, mycetoma, bilharziosis, ankylostomiasis, pellagra, and other diseases which can be studied in the wards. In order to encourage scientific research at the Medical School, the Egyptian Government gives an annual grant of £500 for original work in subjects which possess special importance for Egypt.

We hope to notify the medical and surgical work of the hospital in our next issue. They include an exhaustive article on "The Sclerostomidæ of Horses and Donkeys in Egypt," with thirteen plates by Professor A. Looss. Dr. Wilson contributes an article "On the Poison of Spiders," with especial reference to that of the *Chætopelma olivacea*. Mr. Madden and Mr. Milton give details of several interesting surgical cases, and the former with Dr. Goodman describe "Four Cases of Pinta." Mr. Madden also mentions "Two Cases of the Pink Variety of Mycetoma." Besides these, the table of contents contains "A Case of Foetal Rickets" (Drs. Sandwith and Symmers); "Four Cases of Vaginal Atresia," and "Syphilis in Egypt" (Mr. Madden); "Report on *post mortems* at Kasr-el-Ainy" (Dr. Symmers); "Dietary of the Hospital" (Drs. Sandwith and Wilson); and a valuable compendium of "Statistics of Kasr-el-Ainy," for 1900.

From the above brief enumeration it may be gathered that the visit of the members of the Congress to Cairo will certainly prove both interesting and instructive.

SPECIAL PLAGUE PRECAUTIONS IN INDIA.—The following medical staffs have been appointed to the railway plague inspection stations recently established as a precautionary measure against the inroad of plague on Delhi: Captain J. Hickie, I.M.S., and Miss Schmidt, Umballa Cantonment; Military Assistant-Surgeon W. C. Dicks and Mrs. Dicks, Sonapat; Mr. G. H. Key and Mrs. Convill, Rohtak; Military Assistant-Surgeon W. Charters and Mrs. Dissent, Rewari.

DINNER TO DR. ANDREW BALFOUR,
DIRECTOR OF THE CHEMICAL AND
BACTERIOLOGICAL RESEARCH LABORA-
TORIES OF THE GORDON MEMORIAL
COLLEGE, KHARTOUM.

At Prince's Restaurant, Piccadilly, London, on Monday, December 8th, at the invitation of Henry S. Wellcome, a large party assembled to bid farewell to Dr. Balfour before he leaves for the Soudan, and to wish success to the newly established laboratories.

The guests were received and the dinner was served in the Rooms of the Institute of Painters in Water Colours, which were tastefully and elaborately decorated for the occasion with Egyptian trophies and curios, collected by Mr. Wellcome whilst he sojourned in Upper Egypt.

The guests who responded to Mr. Wellcome's invitation to be present were: Dr. Phineas Abraham, Professor H. E. Armstrong, Professor John Attfield, Dr. Andrew Balfour, Dr. T. J. Barnardo, M. B. Blake, Dr. T. G. Brodie, Dr. Horace T. Brown, J. H. Balfour Browne, Dr. Wallis Budge, Mr. James Cantlie, George Christall, Dr. L. Cobbett, Dr. L. Eliot Creasy, Dr. W. H. Crosse, Dr. C. W. Daniels, Dr. D. S. Davies, Dr. W. Dowson, G. Claridge Druce, Sir Dyce Duckworth, Percy Elford, C. E. Fagan, W. J. Fisher, Dr. A. Chune Fletcher, Dr. Alex. G. R. Foulerton, W. E. Grey, Dr. Alfred S. Gubb, C. Guest, Professor R. Tanner Hewlett, Aubrey T. Hill, Dr. F. G. Hopkins, Prof. G. B. Howes, Sir William Huggins, John Humphrey, H. Finnis Johnson, Dr. H. A. D. Jowett, Prof. R. F. C. Leith, J. M. Le Sage, Edward F. Linstead, Dr. Harvey Littlejohn, Peter MacEwan, Dr. Patrick Manson, John Melanby, John Moore, Dr. Malcolm Morris, Dr. William Murrell, Hon. George Peel, W. G. Piper, M. Post, Sir R. Douglas Powell, Dr. Fred. B. Power, Dr. Joseph Priestley, John Morgan Richards, E. Robbins, Dr. Davies Roberts, A. C. Robinson, A. Gordon Salamon, A. Scott, William Senior, Prof. R. Bowdler Sharpe, Dr. W. Vernon Shaw, Hugh C. Smith, J. Collett Smith, Sir Henry M. Stanley, Prof. E. H. Starling, R. Clay Sudlow, Surg.-Gen. Sir Wm. Taylor, Prof. G. D. Thane, Dr. John C. Thresh, M. Times, Thomas Tyrer, Dr. Thomas Wakley, junr., Maurice Waller, Dr. F. C. Wallis, W. Philip Want, W. Watson-Hill, C. Corning Weld.

Each guest was provided with a handsome booklet containing, not only the *menu* of the dinner, but also "Some Reminders of the Ancient Seats of Learning on the Nile," copiously illustrated.

After the toast of "The King" by the CHAIRMAN, Mr. BALFOUR BROWN, K.C., proposed the toast of "The Rescuers and Administrators of the Soudan" (Lord Kitchener, Lord Cromer, and Sir F. R. Wingate, and others). He said that we had won the Soudan by the sword, but we were now ruling that country and administering it with the scales of justice. The Egyptians may have been struck by our prowess, but they, in common with all races who came under British rule, were more impressed with the honesty and evenly-balanced justice with which they were being ruled.

Sir HENRY STANLEY, G.C.B., LL.D., proposed "Success to the Gordon Memorial College, Khartoum." He remarked that it was but a few years ago since the term "darkest" was wholly appropriate to many regions of Africa, but to-day the advance in civilisation was so pronounced and rapid that light was being admitted to even the most obscure parts. Even in the dark forest, which it had taken him 160 days to struggle through, a new railway to the lakes was actually in course of construction. There was a great future in store for the children of the Soudan, who were the heirs of the ages. They were the descendants of the people who had conquered Egypt and built the pyramids. In fulness of time Africa had come to its destiny. In these remote parts churches were being built, steamers were now plying the river which a few years ago was unknown, railways were multiplying, and given wise rulers, a continuity of policy and steadiness of Government, the people of Africa might attain a high state of usefulness and development. He could imagine no project better calculated to hasten that desirable end than the establishment of a centre of education such as the Gordon Memorial College at Khartoum promised to become. He wished the College all success.

HUGH COLIN SMITH, Trustee of the Gordon Memorial College Fund, in responding to the toast, sketched the foundation of the College from the time Lord Kitchener returned to England after Omdurman. This year, he said, they had not received a report from the head of the College, but last year's report was most interesting. It stated that there were five elementary schools situated in the neighbourhood of the College, where 150 boys were receiving education in order to avail themselves of the advantages of the College when it was opened. The foundation of the College was not merely the centre of the pacification of the country, but also the seat of learning, and nothing could tend so much to the improvement of that country and to the welfare of the population there, than the bringing of the people and the country into touch with the commerce of England.

HENRY S. WELLCOME (the Chairman), in proposing the health of Dr. Andrew Balfour, referred to his own visit to the Soudan, and said that when he was at the College he was struck by the fact that no provision had been made for laboratories, and he recognised the need of research and investigation in that part of the world. He was also struck by the intellect of many of the natives, and he believed that many of them could be trained in the College. He thought it was a splendid chance for some one who would make it a life-work to conduct research out there. They had had many candidates for the directorship, and among them many distinguished men. But there was no one so specially qualified for the work as Dr. Balfour, who had before him the great task of solving problems of research, and thereby conferring a great benefit upon mankind in general.

Dr. BALFOUR, in reply, said it was a splendid device of Mr. Wellcome to give him such a kindly send-off, and to enable him to meet those who were interested

in the undertaking, which was due to Mr. Wellcome's farsightedness, scientific enthusiasm, and generosity. He had always been interested in tropical medicine. He deemed it a great honour to be appointed to the post of director, and he felt that he was about to undertake a great work.

Mr. JAMES CANTLIE, in proposing the toast of "Tropical Medicine," referred to the excellent work that had been done in London and Liverpool by the Schools of Tropical Medicine; and amongst the many students which had passed through the London School they were proud to include Dr. Andrew Balfour amongst the number. He hoped many more would follow the excellent example set by Mr. Wellcome; for we had to look to private citizens to help us to carry out the teaching of Tropical Medicine in this country.

Dr. PATRICK MANSON, C.M.G., F.R.S., in responding to the toast, remarked that they should not be too impatient for the publication of results of Dr. Balfour's work. Real useful work required time and thought, and it could not be hurried. Africa was undergoing an enormous pathological revolution, and the opening up of the country meant, unfortunately, the spread of disease, owing to increased communication. Mr. Chamberlain had grasped the importance of fighting the diseases which overwhelmed many of our richest colonies, and by establishing the London School of Tropical Medicine he had struck at the very root of the means by which the enemy was to be fought. The commission sent out by the Foreign Office and the London School of Tropical Medicine to enquire into the spread of sleeping sickness around the Victoria Nyanza and the upper waters of the Nile had justified their appointment by good work, and he thought it might be interesting to know that they had already discovered the germ and cause of that fell disease.

Sir DYCE DUCKWORTH proposed "Chemical Research," and alluded to the opportunities for extending our knowledge which were opened up by the establishment of chemical laboratories in Khartoum.

Professor H. E. ARMSTRONG, F.R.S., in replying to the toast, stated that in this country there has not been in the past nor was there at present any lack of eminent scientists in chemistry, but that in the application of chemistry to commerce we were undoubtedly behind. Were more of our commercial men endowed with the same spirit as Mr. Wellcome, we would hear less of foreign competition.

Sir R. DOUGLAS POWELL, Bart., proposed the toast of "Bacteriological Research," which was acknowledged by Mr. A. G. R. FOULERTON, Director of Cancer Research Laboratories, Middlesex Hospital.

Mr. A. GORDON SALAMON, Vice-President, Institute of Chemistry, gave the toast of "Science applied to Industries." He aptly illustrated how science became the handmaid of industry, and how the national prosperity might be increased by a close union of the two.

Mr. T. TYLER responded to the toast.

Dr. H. HARVEY LITTLEJOHN, Lecturer on Toxicology, University of Edinburgh, in appropriate terms proposed the "Health of the Chairman," to which Mr. Wellcome briefly responded.

PRESENTATION AND COMPLIMENTARY DINNER TO DR. DANIELS, SUPERINTENDENT LONDON SCHOOL OF TROPICAL MEDICINE.

On November 24th Dr. Daniels was entertained at dinner by the pupils at the London School of Tropical Medicine. Captain Entrican, of the Indian Medical Service, occupied the chair, and there was a full gathering of the students who attended the course. After the usual loyal toasts the Chairman gave "The Guest of the evening, Dr. C. W. Daniels." The Chairman dwelt upon the valuable contributions Dr. Daniels had given to the pathology of tropical diseases, and especially upon his work at the London School of Tropical Medicine. Dr. Daniels had proved himself an eminent teacher; and by his considerate patience and consummate tact, had won the respect and good wishes of not only the members of the present class, but of all the students who have had the privilege and the advantage of coming under his tuition. The contemplated transference of Dr. Daniels to the laboratory at Kuala Lumpur, in the Federated Malay States, for a period of two or three years would be a loss to the school but a gain to science, and it is, he hoped, as he understood it was intended, that Dr. Daniels would return at the termination of his services abroad to once more take up the work of Superintendent of the School. Dr. Taylor Hancock proposed the toast of "The Imperial Forces" in a patriotic speech, which was responded to by Civil Surgeons E. Langley-Hunt, C.M.G., and Lunn. Dr. Sandeman gave the "Colonial Medical Service," which Doctors Cameron Blair and de Boissière acknowledged. Dr. Davies proposed the health of the Vice-Chairman, Dr. Hood, and Dr. Daniels gave the toast of the Chairman.

In the name of the students the Chairman, in an appropriate speech, presented Dr. Daniels with a handsome silver bowl and a micro-photographic apparatus.

As significant of the importance and world-wide influence and reputation of the School, one speaker remarked that the company present was of a cosmopolitan character, no quarter of the globe being unrepresented, but that the different component elements were bound together by a common language, a common flag, and by the educational benefits they had received at the London School of Tropical Medicine.

Current Literature.

SLEEPING SICKNESS IN EAST AFRICA.

C. A. WIGGINS, Medical Officer, East Africa Protectorate, contributes to the *Lancet* notes on sleeping sickness amongst the Wa-Semi, a people belonging to the Wa-Kavirondo residing on the north shore of Kavirondo Bay, Victoria Nyanza Lake. His observations we summarise as follows:—

Advent of the Disease.—Fourteen months previously. *Extent of Infection.*—Half the population.

Etiology.—No *Filaria* *urstanii* parasites were found in the blood of residents in the districts, but

they were found in nearly every Msoga resident on passing through the district, whether he was healthy or otherwise.

Signs and Symptoms.—The expression varies with the stage of the disease. At the end of the first month the expression implies "vacancy"; at the end of the second month the manner becomes listless and vacant; at the end of the third month the saliva dribbles from the mouth, the body is filthy, and the sufferer has to support himself by a stick; at the end of the fourth month the patient lies in his hut without changing his position. Bed-sores are, according to Mr. Wiggins, the chief cause of death. Craw-craw is a common skin complication. An inordinate quickening of the pulse is a constant and special feature of the disease, the average rate during the first month being 116; during the second month, 131; during the third month, 135; and during the fourth month and onwards, 151.

Enlargement of Glands.—The glands, especially the cervical and groin glands, were enlarged in every case of the disease met with; and in children, in six cases, the cervical glands were acutely inflamed.

Sex.—Of 150 cases reported upon, twenty-five only were women, but probably shyness kept the women away.

Duration of Illness.—Two cases only of over five months' duration were seen.

Treatment.—Arsenic was administered, and as much as 20 minims of Fowler's solution were given three times daily to children, with some possible benefit.—*Lancet*, December 13th, 1902.

PLAGUE.

PREVALENCE OF THE DISEASE.

INDIA.—During the weeks ending October 18th and 25th and November 1st and 8th, the deaths from plague in India numbered 10,730, 10,491, 8,915 and 10,488 respectively. Special precautions are being taken in India to ward off plague from Delhi during the approaching Durbar.

EGYPT.—During the weeks ending November 16th, 23rd and 30th, the cases of plague in Egypt were returned as 1, 1 and 0 respectively. The last case of plague occurred in Egypt on November 19th.

CAPE OF GOOD HOPE.—The last case of plague in Cape Colony died on September 23rd; since then there have been no cases of plague in the Colony. Plague-infected rats have been found in Port Elizabeth as late as November 7th.

MAURITIUS.—During the weeks ending November 13th, 20th and 27th, the fresh cases of plague amounted to 23, 30 and 17, and the deaths from the disease to 14, 21 and 12 respectively.

PROPHYLACTIC INOCULATION AGAINST PLAGUE TEMPORARILY SUSPENDED IN THE PUNJAB.—A Reuter's telegram dated Bombay, December 2nd, is responsible for the statements that extensive inoculation against plague has been temporarily suspended in the Punjab owing to the supply of prophylactic serum having given out. The same telegram also states that ten persons died from the effects of the serum used for inoculation having become contaminated; and further,

that the contamination was due to the change suggested by the Plague Commission in the method of preparation. We defer comment upon the part of the communication.

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Archives Russes de Pathologie, de Médec. Clinique et Bacteriologie.
Australasian Medical Gazette.
Boletín de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Brooklyn Medical Journal.
Caducée.
Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Esercito
Hong Kong Telegraph.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
Journal of the American Medical Association.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Medical Record.
Medical Review.
Merck's Archives.
New York Medical Journal.
New York Post-Graduate.
Pacific Medical Journal.
Polyclinic.
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Revista de Medicina Tropical.
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- 5.—Correspondents should look for replies under the heading "Answers to Correspondents."

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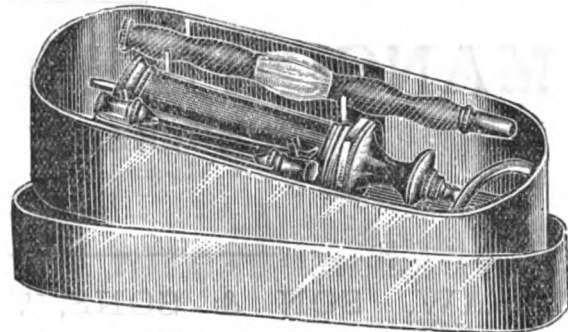
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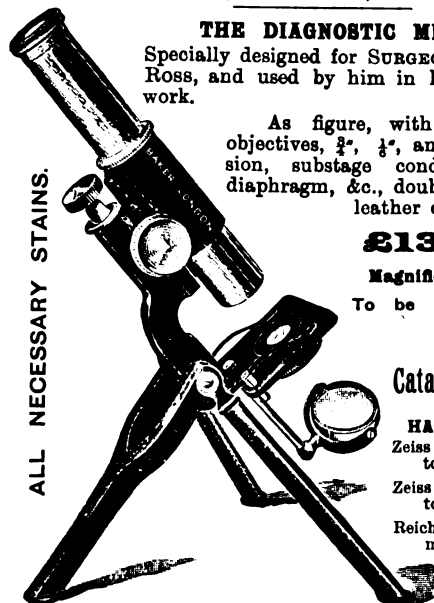
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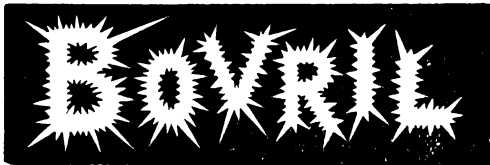
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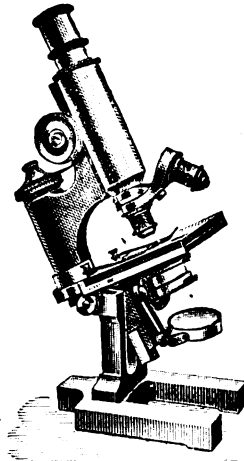
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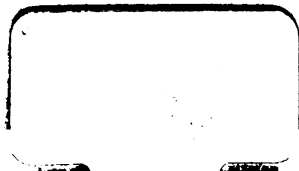
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